

ABSCESS OF THE BRAIN

• • • • •

by

J.B.Pennybacker, M.A., M.B., Ch.B., F.R.C.S.

Oxford, 1944.



FOREWORD

.....

I feel that some explanation should be offered in submitting a thesis on Abscess of the Brain for the M.D. degree. Although it is generally regarded as a surgical subject, it will be seen that the various technical procedures constitute a very small part of the problem, and that general medical principles govern diagnosis and all of treatment except the operation itself. I would say that of the worry and difficulty in dealing with these cases, not more than five per cent has to do with the actual operation - for the rest, a physician would be as much concerned as a surgeon.

Neurosurgery is a rather hybrid specialty, and for those who practice it a sound knowledge of general medicine and neurology is just as necessary as operative competence. Indeed, in our own University we have a Department of Surgical Neurology rather than a department of neurological surgery. The slight shift of emphasis is a significant development because for many years diseases have been losing their identity as purely "medical" or "surgical" problems. I considered that Abscess of the Brain was a sufficiently good example of this equal concern for physician and surgeon to justify the presentation of this thesis.

Oxford, 1944.

C O N T E N T S

.

Introduction	page 1.
Aetiology	5
Pulmonary Infections	7
Infective Endocarditis	9
Metastases from Other Sources	9
Mastoid Infections	12
Frontal Sinusitis	17
Penetrating Wounds	21
Infections of Face, Scalp and Skull	27
Pathology	31
Bacteriology	36
Symptoms and Signs of Brain Abscess	39
Mode of Onset	43
Constitutional Symptoms	52
Examination of the Cerebrospinal Fluid	61
Radiology in Diagnosis	67
Neurological Signs	72
Frontal Lobe	75
Temporal Lobe	79
Parietal Lobe	84

Symptoms and Signs of Brain Abscess	page 39
Neurological Signs	72
Occipital Lobe	87
Cerebellum	89
Differential Diagnosis	96
Other Expanding Lesions	97
Intracranial Venous Obstruction ("Otitic Hydrocephalus", cerebral thrombo- phlebitis, etc.)	100
Other Intracranial Infections	105
Extradural Abscess	105
Subdural Abscess	106
Leptomeningitis	108
Tuberculous Meningitis	110
Petrositis	111
Arterial Hypertension	112
Psychoneuroses	113
Treatment	114
Priority of Treatment	150
Test of Cure	152
Late Results	154
Bibliography	159
Case Reports : index Cases 1-20	160

I. Introduction.

Fifty years have passed since the publication of Macewen's book on "Pyogenic Diseases of the Brain and Spinal Cord" (1). Viewed at a distance of half a century, it remains a landmark because up until this time abscess of the brain had been regarded as almost invariably fatal. Gull (2) in 1857 reported a series of cases in which the possibility of treatment or of any other than a fatal outcome was apparently not considered, the chief interest at that time being in the morbid anatomy and diagnosis of the disease. From then until 1893 there were isolated reports of cases treated surgically: indeed, prior to 1850 Dupuytren and Detmold had drained cerebral abscesses; Hitzig recorded a case in 1871 accurately localised in the posterior part of the left frontal lobe before his experiments on cortical representation. Sajou's Annual from 1888 to 1892 mentions 55 abscesses of the brain which had been dealt with by operation (3). But the tenor of all these reports reflects the dramatic aspect of the cases which ended as triumphs for a bold surgeon or more often simply as matters of pathological and clinical interest. Then came Macewen, who summarised his experience of twenty-five abscesses of the brain, of which nineteen were operated on and eighteen recovered, and

concluded "that in uncomplicated abscess of the brain operated on at a fairly early period, recovery ought to be the rule".

In the same year a physician, Allen Starr of New York, published the first book in the English language on "Brain Surgery" (4) and included a chapter on the operative treatment of abscess of the brain. Macewen's results were apparently not known to him, and the fact that these two books appeared in the same year in Europe and America bespeaks the wakening interest in the possibility of treating successfully a hitherto fatal disease. A number of factors contributed: it was the age of dramatic surgery, already emboldened by developments in anaesthesia and asepsis and already looking for new worlds to conquer. But, even more important, it was the golden era of neurology: the work of Fritsch, Hitzig, Ferrier, and Hughlings Jackson had revealed something of the workings of the brain and had established the basic facts of cerebral localisation. The first two decades of the twentieth century saw important advances in our knowledge of the circulation of the cerebrospinal fluid and intracranial hydrodynamics. When lumbar puncture became a common practice, examination of the cerebrospinal fluid assumed a new importance in the investigation of infections of the nervous system.

Radiology was making rapid progress from the stage of an interesting physical phenomenon to a common clinical practice, and the introduction of ventriculography opened a new era in neurological diagnosis. These advances went hand in hand with the work of Cushing, who was showing that brain surgery was a practical proposition and was training his pupils in a technique which was to be practised throughout the civilised world.

Despite these advances, the treatment of brain abscess* continues to be a serious problem, and a survey of the literature is a depressing study. Elkington (5) in 1939 found that the operative mortality in various American and British clinics varied between 41 per cent. and 79 per cent. No surgeon today could express Mac-ewen's conclusion as other than a pious hope, and few approach a case of brain abscess at whatever period with any degree of confidence in the outcome. The present study began with a primary concern for the unsatisfactory state of therapy, but it was soon apparent from a review of the literature that there is still much to be learned about diagnosis, and that in no small part the mortality of brain abscess is due to the diagnosis

* The term "brain abscess" is used throughout to include both cerebral and cerebellar abscesses. It does not include other pyogenic processes such as extradural abscess, subdural abscess, and purulent meningitis.

being made too late or not being made at all. The subject is of wide practical importance because a brain abscess may arise from disease in almost any part of the body, and as the responsibility of diagnosis may rest on any general practitioner or specialist it seems that a review in the light of present knowledge should be of some value.

The clinical material comprises some 50 consecutive cases of brain abscess seen in the Nuffield Department of Surgery, Oxford, during a six-year period from May, 1938. Some of the cases were referred directly to the Department as suffering from increased intracranial pressure; the others were drawn from the general medical and surgical wards and from the Ear, Nose and Throat Department of the Radcliffe Infirmary, Oxford. The material is thus fairly representative, but there are limitations - e.g., the paucity of abscesses due to gunshot wounds of the head, a group which does not figure largely in civilian practice in this country, but which may be expected to demand more attention in the immediate future as the result of battle casualties.

All of the patients who survived have been followed up, and not the least interesting part of the study has been to see the late results of a massive infection and major operation on the brain. With some particularly interesting psychological sequels I was fortunate in

having the advice and opinions of the late Professor Kurt Koffka, Dr. Erich Guttman, and the social workers associated with them. I was also fortunate in seeing and following at first hand the researches of Drs. Russell, MacFarlan and Falconer on the experimental production of brain abscess. Throughout the study I had the advice and encouragement of Professor Hugh Cairns and the invaluable assistance of a succession of residents whose ability and enthusiasm contributed largely to the results.

II. Aetiology.

An abscess may result from infection which reaches the brain either by direct extension (e.g., from the mastoid), by implantation as in penetrating wounds, or by the blood stream. There is no "primary" or "idiopathic" brain abscess: as with other morbid processes, to use such a term simply means that we have not succeeded in identifying the source, which may have been a forgotten boil or septic skin spot, or a small focus of infection in other parts of the body which caused no remarkable symptoms.

The following table shows the origin of the 50 cases dealt with in this study.

TABLE I. AETIOLOGY OF 50 CASES OF BRAIN ABSCESS.

Source of Infection	No. of Cases	Died
I. Metastatic	16 (32%)	9
A. From pulmonary infection (empyema, lung abscess, bronchiectasis)	7	6
B. From infective endocarditis	2	2
C. From other sources (furunculosis 4; cellulitis 1; tuberculosis 1; not known 1)	7	1*
II. Mastoid Infection	18 (36%)	10
A. Cerebral abscess (temporal lobe)	11	4
B. Cerebellar abscess	7	6
III. Frontal Sinus Infection	6 (12%)	0
IV. Penetrating Wounds	4 (8%)	1*
V. Infections of Face, Scalp & Skull (Infection of tumour cavity 2; abscess of eyebrow 1; carbuncle of scalp 1; boil on cheek and cavern- ous sinus thrombosis 1; infection of antrum and osteitis of skull 1)	6 (12%)	1*
TOTAL	50	21 (42%)

* Undiagnosed and untreated.

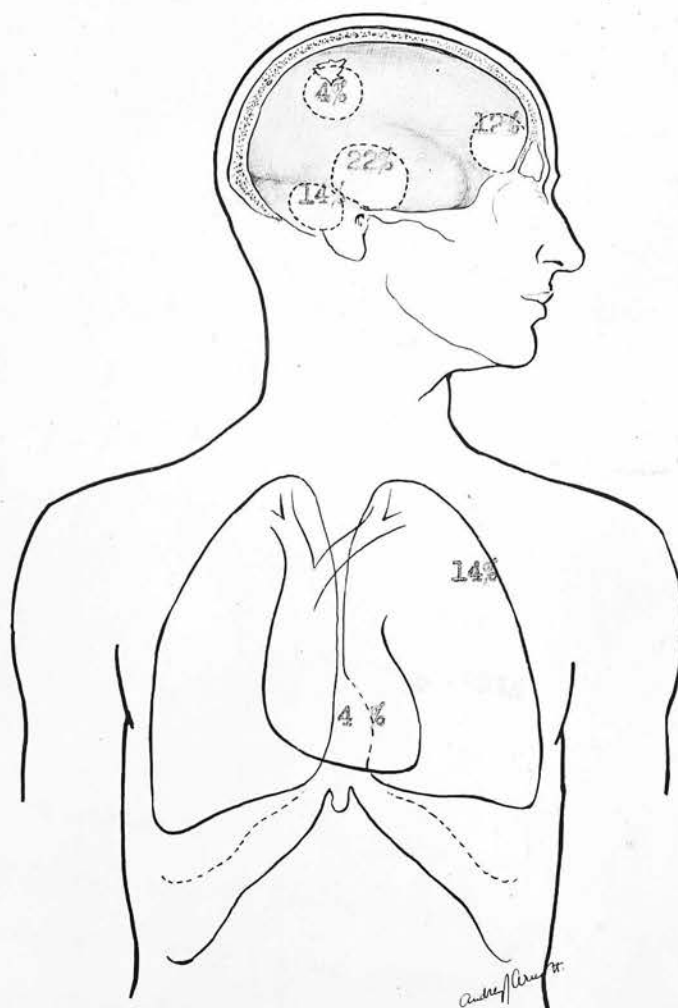


Fig. 1 - Aetiology of 50 Cases of Brain Abscess.

Pulmonary Infection	14%
Infective Endocarditis	4%
Metastatic from Other Sources	14%
Mastoid Disease	36%
(Temporal 22%	
Cerebellar 14%)	
Frontal Sinus Infection	12%
Infections of Face, Scalp, and Skull	12%
Penetrating Wounds	8%

The distribution set out above is in general agreement with other reports, but as all of these cases occurred during the sulphonamide era it is interesting to compare this analysis with an earlier series reported by Evans (6) in 1931. Of 194 brain abscesses found at autopsy 62 per cent. were due to infections of the mastoid and paranasal sinuses. In the Oxford Series these groups comprised 48 per cent. This accords with a general impression that the incidence of chronic mastoiditis has been lessened by modern chemotherapy, and it is probable that further advances will lessen still more the serious intracranial complications. It is likewise not too much to hope that the incidence of chronic pulmonary sepsis may be lessened by improvements in public health measures and advances in chemotherapy for acute infections: at present the brain abscesses due to these infections are a particularly sombre group, and any advance in prevention should be welcomed.

Abscesses due to Intrathoracic Infection. Gull (2) in 1857 first drew attention to the frequency with which brain abscess develops as a complication of lung sepsis. Since then the association has become so familiar that the diagnosis is rarely missed. In the vast majority of cases the pulmonary infection is a chronic one: we have encountered only one case of brain abscess following an

acute empyema which had responded to treatment and no cases due to acute lobar pneumonia which had resolved. The cerebral infection may be simply an incident in the course of a chronic illness, as in Case 1; or it may occur like a bolt from the blue in a person with mild chronic bronchiectasis who is otherwise in good health, as in Case 2; or it may occur during a mild respiratory infection superimposed on chronic bronchiectasis, as in Case 3. In one case (Case 4) the first manifestation of cerebral infection occurred several weeks after an isolated lung abscess had resolved by rupturing into a bronchus.

The infection becomes blood-borne by dislodgement of an infected thrombus or by the erosion of a small blood vessel in a purulent zone. It would be expected that the necrotising lesions (e.g., lung abscess and chronic empyema) would behave in this way more commonly than the acute infections. Having gained the blood stream, the organisms are carried to the left heart by the pulmonary veins, thence into the general arterial circulation. It is remarkable that in almost all cases the infection reaches the brain by way of the internal carotid circulation, the abscess developing in the distribution of the middle and anterior cerebral arteries, but I have recently seen a case in which presumably it

was carried by the vertebral artery: a middle-aged man with chronic bronchiectasis was suddenly rendered blind in both eyes, the blindness having the features associated with a bilateral occipital lesion. At the autopsy abscesses were found at the posterior end of both occipital lobes (Fig. 2). That they occurred simultaneously suggests that the embolus had travelled by way of the basilar artery and had lodged in its two terminal branches, the posterior cerebral arteries.

Abscess of the brain due to infective endocarditis is probably not as common as the incidence in this series would suggest. The interest attaching to these two cases is that in one the brain abscess was the only focus of suppuration outside the heart; and in the other (Case 5) the brain abscess was the first embolic phenomenon to be detected and it attained such a size as to endanger life before other manifestations of the disease were recognised.

The third group of metastatic abscesses comprises those from "other sources". In some cases the primary infection may not be readily identified, and the possibilities which have to be borne in mind are boils, carbuncles, septic skin spots (Case 6), dental abscesses, etc. It is worthy of note ~~that~~ these abscesses of doubtful origin are usually single ones and in a fairly chronic stage by the time they are presented for treatment; indeed,

MULTIPLE BRONCHOGENIC ABSCESSSES.

Note in Fig. 2 abscesses in occipital lobes, and in Figs. 3 and 4 the tendency for the lesions to occur at the junction of grey and white matter. The abscess in the right basal ganglia seen in Fig. 5 is a rare exception to the general rule that the depths of the brain escape metastatic abscess formation. The swelling of the thalamus seen in Fig. 4 is due to oedema around the abscess.



Fig. 2.

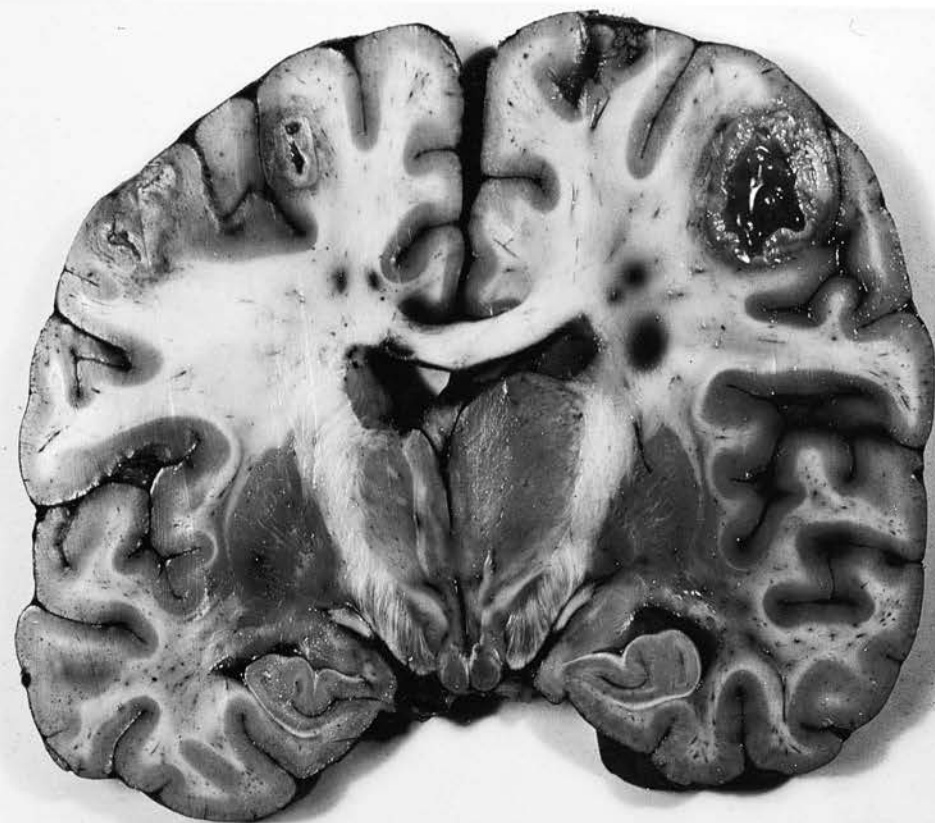


Fig. 4.



Fig. 3.

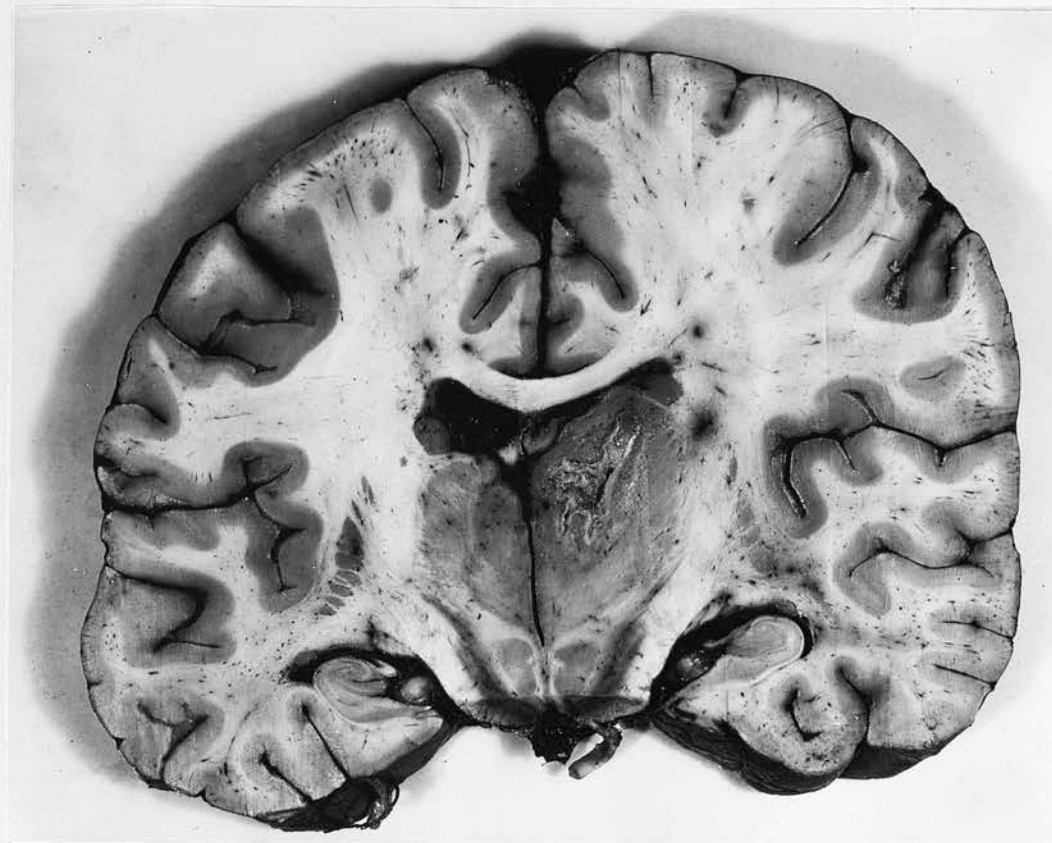


Fig. 5 .

the primary infection may have been so trivial or so remote in time that its relation to the intracranial lesion is forgotten. In such cases the lesion may be regarded as a neoplasm until it is disclosed at operation. In one case (Case 7) the course of the infection remained a mystery. These single chronic abscesses are to be contrasted with those due to lung sepsis which have their origin in showers of infected emboli and are usually multiple; and occurring as a complication of a debilitating disease they usually run a very acute course. Case 4 is a rare exception to this generalisation.

Cameron (7) in 1907 drew attention to a difference between brain abscess due to pulmonary suppuration and those due to pyemia from other sources: in the former, the brain abscess is usually the only focus of suppuration outside the thorax, whereas in pyemia the cerebral infection may be only part of a picture of multiple abscesses scattered throughout the body, in the liver, spleen, kidneys, etc. In this series we have been struck by the rarity of any other than a cerebral infection in both groups. This suggests that, despite its considerable blood supply, the brain is less well equipped to deal with infection than other parts of the body, and that once an infection becomes established the chances of spontaneous resolution are small.

Metastatic abscesses may be found in any part of the brain, but as with blood-borne neoplasms they often begin at the junction of white and grey matter (Figs. 3 - 4) and it is remarkable how rarely they are found in inaccessible sites such as the basal ganglia, corpus callosum, brain stem, etc. (But see Fig. 5.) The problem of treatment is thus not so much one of the accessibility of the abscess as of the nature of the primary disease. It will be seen from Table I that there is a striking difference in the mortality rate of the sub-groups: of 9 thoracogenic abscesses 8 died, whereas only one of the 7 patients in the third group ("other sources") died. The chief factor governing this difference is that in the case of thoracogenic abscess the cerebral infection is a complication of continuing sepsis, and treating such an abscess is in some respects analagous to dealing with a cerebral metastasis from a primary carcinoma of the lung: in Case 1 the abscess in the right cerebral hemisphere was dealt with successfully, but the continuing chest disease later gave rise to further abscesses in the left hemisphere, one of which proved fatal. The same consideration applies to the abscesses due to infective endocarditis and renders them hopeless in the light of present knowledge, as no cure for the primary infection is known. In the third group, on the other hand, the primary infection has

usually resolved completely by the time the brain abscess demands treatment, and the problem is more simply that of dealing with an unresolved sequel.

There is another important factor which contributes to the mortality of abscesses due to chronic pulmonary disease. The stupor and respiratory depression caused by the brain abscess mean that the purulent exudate is not coughed up from the lungs, and it is common for these patients to die drowning in their own purulent sputum. Swallowing may be difficult and the risks of inhalation pneumonia are considerable too. Although, as shown in Case 4, there is nothing essentially fatal about a bronchogenic abscess, the present situation is so grave that prevention and treatment of chronic pulmonary sepsis seems to be the most promising line of attack on this part of the problem.

Brain Abscesses due to Mastoid Infection. Mastoid disease is the commonest single cause of brain abscess. In this series it was responsible for 36 per cent. of the cases, although in earlier reports the proportion is generally higher, e.g., Evans (6) 56 per cent. The modern view is that every infection of the middle ear cleft involves the mastoid air cells, and it is worthy of note that a brain abscess may develop at any stage of such an infection. Thus, as in Cases 8 and 9, an acute

middle-ear infection may run its whole course in two or three weeks, subside without radical treatment, and yet a brain abscess results. More commonly the abscess is a complication of chronic disease, the brain abscess developing insidiously in a patient with a chronic discharging ear, or calling attention to itself by a flare-up of a chronic infection ("acute-on-chronic" mastoiditis), as in Case 10. Such flare-ups may betoken nothing more than a local infection in the mastoid, however, or they may mean the formation of an extradural abscess (as in Case 11) proceeding to a spread of the infection to the brain itself. Other pathological possibilities not falling into the purview of this study are subdural abscess, thrombosis of the lateral sinus, and diffuse leptomeningitis.

The brain abscesses due to mastoid disease are either in the temporal lobe or in the cerebellum. There seem to be two routes by which the infection may spread. A vein develops an infected thrombus which extends along the vein through the dura and the leptomeninges into the substance of the brain, where a focus of suppuration starts. Connective tissue changes around the infected vein may form a more or less definite stalk tethering the abscess to the dura. Although the pathological evidence is insufficient, it is my impression that the formation of a stalk is more likely to occur in abscesses due to acute middle-ear infections which resolve without bony involvement,

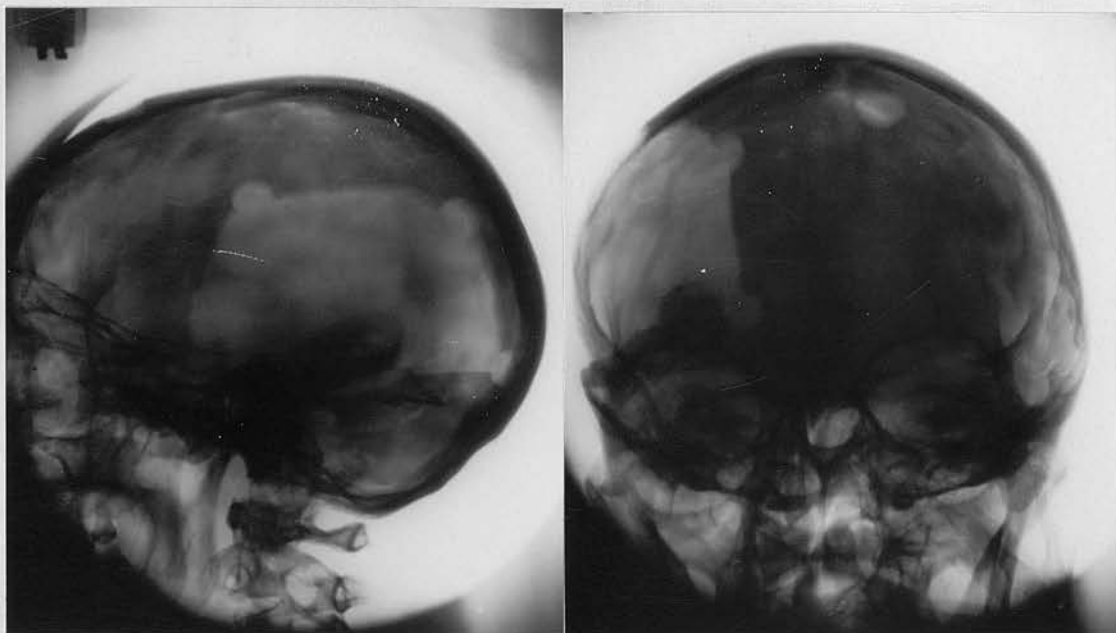
as in Case 8. More commonly there is infection of the bone in contact with the dura, sometimes actual necrosis, e.g., of the tegmen tympani. A localised meningitis ensues leading to an adhesion which forms a broad sessile attachment of the abscess to the dura. Again, there is little opportunity of demonstrating these processes, but the broad attachment of the abscess to the dura is a common finding in cases of chronic mastoiditis, in which the spread of the infection to the brain may be said to be a process of extension by continuity. By whatever route the infection reaches the brain, it should be noted that any of the tissue planes which it traverses may become infected, leading to the formation of an extradural abscess, purulent pachymeningitis, subdural abscess, or diffuse purulent leptomeningitis.

The position of the temporal lobe*abscess is fairly constant. The sessile attachment or the stalk is usually in the region of the tegmen tympani, and the abscess expands in the temporal lobe to form an ovoid mass which displaces the Sylvian fissure upwards and the inferior horn of the ventricle medially (Fig. 26 p. 139). The wall of the abscess usually abuts on or is actually adherent to

* These abscesses are usually referred to by otologists as "temporo-sphenoidal", but as this term has no special descriptive advantage over the simpler "temporal" it should be discontinued.

the wall of the ventricle, and when such an abscess is removed at operation it is often necessary to excise a portion of the adherent ventricular wall (Fig. 26 p. 138), thus throwing the ventricular system open to the cavity in the temporal lobe from which the abscess has been removed. Occasionally a temporal abscess may drain intermittently into the middle ear: in one case in this series there was a profuse discharge of pus from the external auditory meatus necessitating clean dressings three or four times a day. When the chronic abscess was excised from the temporal lobe the discharge ceased abruptly and never recurred.

We have recently encountered two cases of cerebral abscess due to mastoiditis in which the abscess was not found in the situation described above - i.e., in the middle of the temporal lobe - but was situated farther back, in the posterior part of the temporal lobe in one case and in the occipital lobe in the other (Figs. 6 - 7). In each case the dura had been pierced during the mastoid operation, in search of a temporal abscess, and in each case a fungus cerebri subsequently developed in the mastoid wound (Fig. 8). It seems probable that the infection was implanted by the exploring instrument introduced through the infected mastoid wound (see p. 151), as we have only encountered this variation from the usual



Figs. 6 and 7. Pyelograms in a case of right temporal abscess, due to acute mastoiditis. The abscess was not found in the usual position when an attempt was made to aspirate it, but it was found further posteriorly. There was in addition a subdural abscess which had tracked from the mastoid wound (bearing a fungus cerebri) under the occipital lobe, and it is probable that the intracerebral abscess arose from this subdural abscess rather than directly from the mastoid through the tegmen tympani as is usually the case. The outline of the subdural abscess can be seen below and behind the oval temporal abscess.



Fig. 8. Fungus cerebri occurring through a mastoid wound. The dura had been penetrated in a search for pus, but nothing was found and within a few days the fungus made its appearance. Subsequently the child was found to have a large subdural abscess, which behaved in much the same manner as that described in Figs. 6-7, producing a large abscess in the temporo-occipital part of the brain.

position in these two cases.

The cerebellar abscesses are likewise in a fairly constant position: the dural attachment is on the posterior surface of the petrous bone in the region of Trautmann's triangle, and the abscess expands in the anterior part of the lateral lobe of the cerebellum, displacing the fourth ventricle and the brain stem to the opposite side. There is an occasional variation: in one case in this series a cerebellar abscess was suspected and aspiration in the usual site was attempted, but the abscess was not located. At the autopsy it was found that the mastoid infection had caused a purulent thrombosis of the lateral sinus and this infection had spread along one of the tributary veins from the vermis cerebelli to cause an abscess in that situation.

It will be seen from Table I that next to thoracogenic abscesses (of which 8 out of 9 were fatal) those due to mastoid infections carry the highest mortality. Four out of 11 temporal abscesses and 6 out of 7 cerebellar abscesses ended fatally, a mortality of 55 per cent. The facts of location set out above contribute to the hazards of these abscesses. Those in the temporal lobe, being in such intimate relation to the ventricle, can easily rupture into the ventricle and cause an overwhelming infection of the nervous system for which present treatment is of no avail. Furthermore, an expanding lesion in the

temporal lobe quickly produces herniation of the uncus through the incisura tentorii with as dangerous hydrodynamic effects as the better-known herniation of the cerebellar tonsils through the foramen magnum. In the cerebellum an abscess may cause not only tonsillar herniation, but also an upward herniation of the superior surface of the cerebellum through the incisura tentorii (Fig. 40 p212). The nearness of the abscess to the brain stem renders these patients liable to the rapid onset of medullary compression or oedema which may be fatal within a few minutes, as in Case 13.

Brain Abscess due to Frontal Sinus Infection. Infection of the frontal sinus accounted for 12 per cent. of the cases in this series. In every case it was an acute infection of the sinus, accompanied by oedema, redness and tenderness of the forehead on one or both sides. These signs are usually interpreted as meaning the extension of the infection from the mucous membrane to the bony wall of the sinus. If that is so, such an osteitis may resolve spontaneously, as in most of the cases in this series the symptoms and signs of frontal sinusitis and osteitis had subsided without radical treatment by the time the brain abscess was manifest. In only one case (Case 14) was external drainage of a frontal empyema established, and although there were symptoms of

an intracranial infection during this stage the sinus infection had resolved by the time the brain abscess demanded treatment.

Another possible explanation for the superficial signs of inflammation described above is that they are due to infected thromboses of veins passing from the sinus to the scalp superficially and, by inference, to the meninges and brain deep to the sinus. This brings the spread of infection into line with the processes suggested above to explain the intracranial extension of mastoid disease. Again, pathological evidence is scanty, but operative observations on cases of acute frontal sinusitis without intracranial symptoms or signs suggest that bony infection is probably the commoner mechanism: in such cases it is common to find inflammatory changes in the bone, especially of the posterior wall of the sinus, and there is often a button of granulation tissue between the dura and the posterior wall of the sinus, as is frequently seen in osteitis of the vault of the skull due to other causes. This infected granulation tissue offers a means of spread to the brain, and if an abscess develops it will be adherent to the dura at this site. In one case in this series the frontal sinusitis subsided, but was followed by a low-grade osteitis of the adjacent frontal bone which in turn was followed by a brain abscess.

The fact that the frontal sinus is so much less complex than the mastoid doubtless contributes to the comparative ease with which an acute infection subsides without radical treatment. However, that a cerebral abscess may start during an acute infection and continue to enlarge after the sinusitis has resolved may lead to diagnostic difficulties: by the time the abscess produces symptoms or signs noticed by the patient the sinusitis may have been forgotten or dismissed as irrelevant to his present trouble. In one case in this series an interval of several weeks between the sinus infection and the onset of serious intracranial symptoms led to a mistaken diagnosis of an inoperable tumour, and by the time the correct diagnosis was made the patient was totally blind from secondary optic atrophy.

The abscesses due to frontal sinus infection are in the anterior part of the frontal lobe (Fig. 41 p. 216). They are usually adherent to the dura over the posterior aspect of the frontal sinus, but their position varies with the extent and development of the sinus and the part of the bony wall through which the infection has spread. Thus in one case the attachment was in the medial part of the orbital plate and the abscess occupied a nearly mid-line position in the anterior fossa (Fig. 9). In other cases the attachment may be higher in the vault of the skull: this may happen when the disease spreads to the adjacent

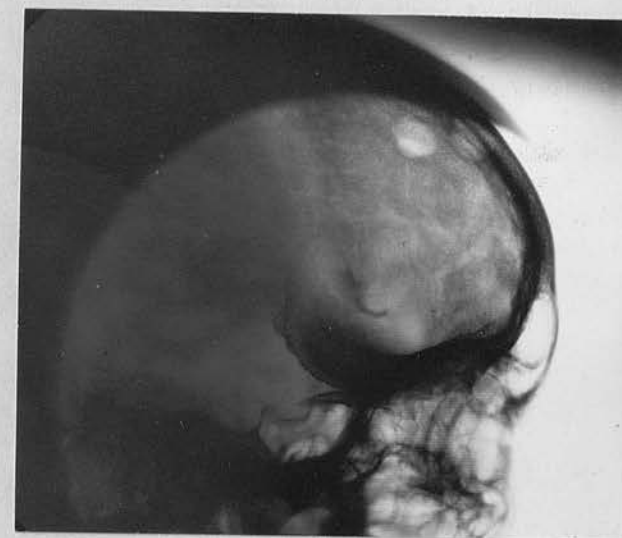
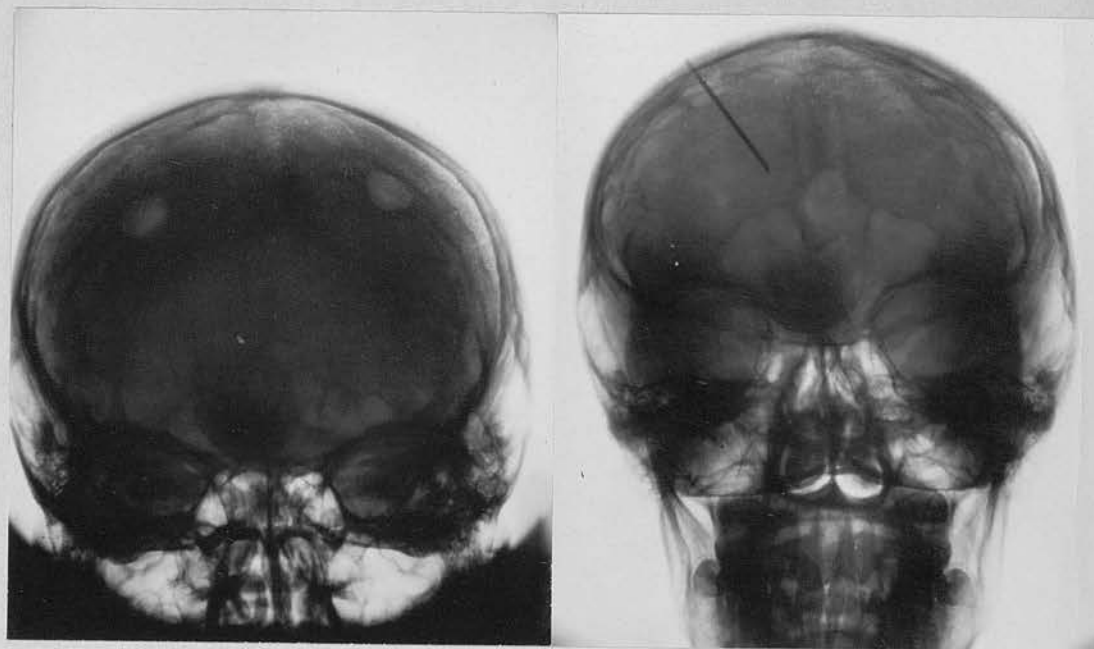
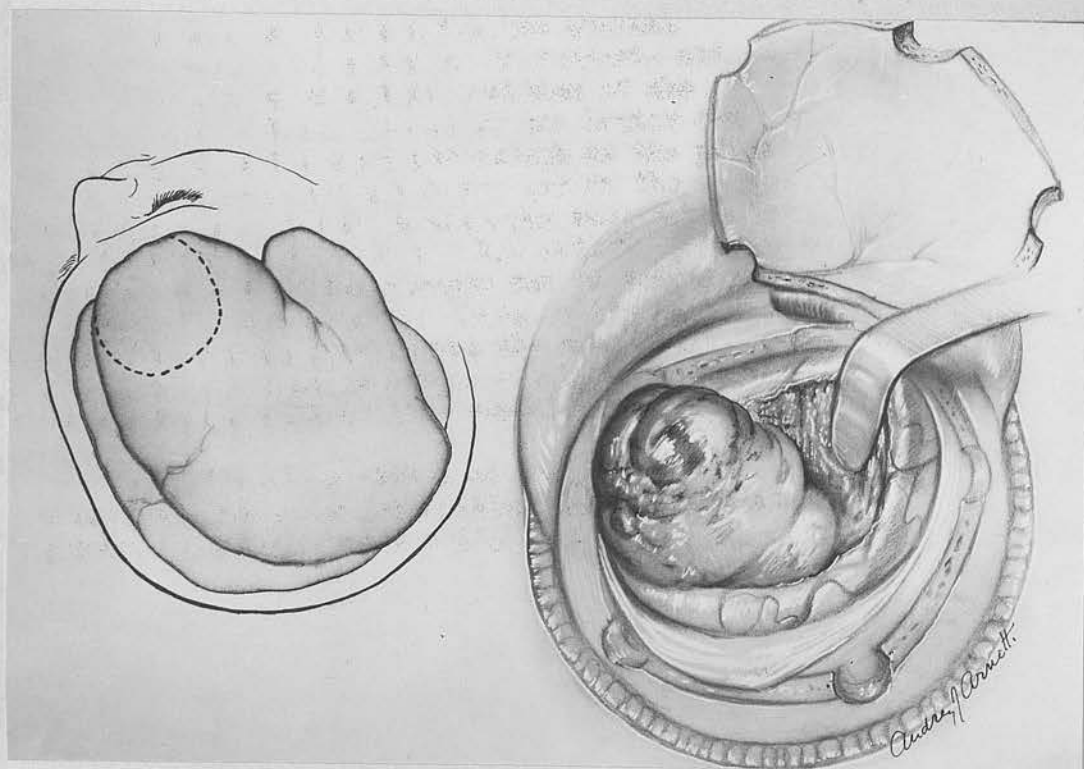


Fig. 9. Abscess due to frontal sinusitis. The upper left photograph was taken after the initial aspiration and instillation of thorotrast, and shows the nearly middle-line position of the abscess. The vertical extent of the lesion can be inferred from the next photograph as the point of the aspirating needle was engaged in the abscess cavity. These photographs were taken at an interval of two weeks. The antero-posterior extent of the abscess can be judged from the lateral projection above as its posterior margin can be seen overhanging the sphenoidal ridge and its anterior margin was known to be adherent to the back of the frontal sinus.

The operation sketch at the left shows the approach used for extirpation and the medial attachment of the abscess wall.



frontal bone (frontal osteitis) and the cerebral infection proceeds from this extension rather than from the primary infection of the sinus.

Table I shows that the operative results in this group are more favourable than in any other: all of the 6 patients survived. The position of the abscess doubtless contributes to the results: an expanding lesion at the frontal pole is at some distance from the ventricular system and the indirect pressure effects due to herniation are less hazardous than those due to lesions in the temporal lobe and cerebellum. Furthermore, abscesses in the frontal lobe are easier of access for whatever method of treatment than those due to mastoid disease.

It has been stated above that all the cases in this series were due to acute infections of the sinus. We have not encountered an abscess due to chronic sinusitis, although there is no reason why they should not occur. In our experience chronic frontal sinusitis is only one aspect of pansinusitis involving the ethmoid, sphenoid, and maxillary air sinuses too. This may lead to an extensive osteitis of the base of the skull or of the facial bones, with purulent infiltration of the soft tissues. The infection may spread to the cranial cavity, but if it does so it is usually as an extensive subdural abscess or purulent pachymeningitis which proves fatal.

In only one case (Case 9) in this series was an infection of the maxillary antrum related to a brain abscess, but the relation was an indirect one, as described in the previous paragraph. This patient had had a mild chronic infection of the right antrum which caused no serious trouble until the floor of the antrum was fractured during a dental extraction. He then got a spreading osteitis of the facial bones and base of the skull on the right side, extending up to the right squamous temporal and the lower part of the adjacent frontal bone. There was purulent infiltration of the temporal muscle, with superficial signs of inflammation. When the infected bone was removed an extradural abscess was found over the temporal pole and there was also an abscess within the temporal lobe beneath the extradural collection. Probably none of this would have happened had the antrum not been fractured, but likewise it would not have happened had the antrum not been infected.

Abscesses due to Penetrating Wounds. Penetrating wounds of the skull and brain accounted for only 8 per cent. of the cases in this series, but the head injuries of war will probably be responsible for a much greater incidence of this type of abscess in the immediate future. One case was due to a gunshot wound of the occipital lobe; one to penetration of the frontal lobe by a sharp

instrument (Case 15); and two to compound fractures of the skull with indriven fragments of bone (Case 16).

A penetrating wound may carry an infected fragment (of missile, bone, clothing, hair, scalp, etc.) into any part of the brain, but it is remarkable how often such objects may become lodged in the brain and yet not cause an abscess. The risks are enhanced if there is superficial sepsis in the wound, as in Case 15, and as in the single case of abscess due to gunshot wound.

This was a soldier who received a shrapnel wound in the left occipital region during the evacuation of Dunkirk on 1st June, 1940. When he was examined ten days later the wound was suppurating and X-rays showed a metallic foreign body in the left occipital lobe, with a small perforation of the skull overlying it. The only neurological abnormality was a lower-quadrant hemianopia in the right homonymous field of vision. The scalp wound was opened and drained and it healed by the middle of July, 1940. He was free from symptoms except for an occasional pain at the site of the wound and the persistent defect in the visual field. The cerebrospinal fluid was still abnormal in containing 68 mgm. protein and 17 cells. This suggested the presence of a brain abscess, but an encephalogram showed a slight ventricular dilatation without any local deformity; it was thought that this

excluded the possibility of an abscess, and he was discharged from hospital. He resumed work on 1st November, 1940, and carried on in fairly good health until August, 1942, when he developed a paritonsillar abscess which was accompanied by a good deal of headache. He recovered from this and resumed his work as a bus-driver. On 2nd October, 1942, he was suddenly stricken with the signs and symptoms of a low-grade meningitis. This did not respond to the usual chemotherapeutics, so he was sent to hospital on 7th October. Although he complained of severe headache, he was quite alert and rational. On the same day, shortly after admission, he suddenly said that he could not move his legs, and he died within a few minutes - 2 years and 5 months after the original injury. At the autopsy **the** scalp wound was soundly healed and the hole in the bone was occupied by a firm, fibrous scar. The shell fragment was embedded in a glial scar in the upper part of the occipital lobe and abutting on to this scar there was an oval abscess measuring 3.5 x 2 x 4 cm. from before backwards. The antero-inferior wall of the abscess was in contact with the ventricle. The abscess had a well-defined capsule up to 0.3 cm. thick. There was a diffuse purulent meningitis and ventriculitis which doubtless was responsible for death. A haemolytic streptococcus was grown from the pus in the abscess and the same

organism was recovered from the meninges and ventricles.

In a large series of gunshot wounds of the head treated during the Libyan campaign of the present war Ascroft (8) found that retained metal was on the whole less likely to cause a brain abscess than retained bony fragments, and he stresses the importance of removing all such fragments from the track of the missile in the primary treatment of the wound. It is still too early to make a final assessment of the risks of retained metal fragments, because of the remarkable chronicity of the abscess formation: in the case cited above $2\frac{1}{2}$ years elapsed between the injury and death, and some cases of gunshot wound in the first World War were followed by abscesses which did not demand treatment until 15 years later. More commonly, of course, the abscess develops quickly within a few weeks of the injury; and in some cases the infection reaches the ventricular system and causes death before a localised abscess has had time to develop. This is a frequent cause of death in gunshot wounds of the head.

In the two cases of compound fracture of the skull in this series the dura and underlying brain were lacerated, and presumably the infection was implanted at the time. However, a brain abscess may result from an infected compound fracture even though the dura is intact: such cases are essentially similar to those of osteitis of the

skull due to other causes and they will be discussed in the next section.

The abscesses due to compound fracture and penetrating wounds are apt to be associated with the formation of a brain fungus. The two factors which contribute to the development of a fungus are (1) increased intracranial pressure; and (2) a defect in all of the coverings of the brain (scalp, skull, and dura) over the same area. These two factors may be somewhat dependent on each other: an untreated wound which destroys the scalp, skull and dura and exposes the brain is almost always followed by inflammation of the exposed brain. This raises the intracranial pressure and tends to force the brain out of the cranium through the defect in the coverings. Moreover, even if the scalp has been sutured over a defect in the skull and dura - e.g., in the repair of a compound fracture - infection of the brain may cause herniation through the skull defect which bursts the scalp wound and allows the brain to present on the surface as a fungus. The formation of a fungus is usually accompanied by dilatation of the underlying ventricle, and O'Connell (9) in a recent study suggests that this ventricular dilatation is the most important causative factor in fungation, but the evidence is incomplete and it is at least as likely that the dilatation is a secondary effect of the

fungus. Ventricular dilatation is a common occurrence in cerebral hernia due to various causes, and a fungus is essentially a hernia which is not covered by the scalp.

A fungus usually develops within ten days or a fortnight of the injury. The exposed brain loses its characteristic appearance, being covered by necrotic tissue and inflammatory exudate if its surface has been torn, and obscured by intense congestion of the leptomeninges if its surface is intact. Gradually it assumes an even red colour and a granular surface due to the formation of granulation tissue. If healing takes place, it does so by concentric epithelialisation, leaving a firm scar composed of skin, fibrous and glial tissue which anchors the brain securely at this point. When epithelialisation is complete, the fungus reverts to the status of a hernia. The persistence of a hernia bespeaks a continuing increase in intracranial pressure, and in cases of head injury the most common cause is an abscess of the brain. On the other hand, there are cases of brain fungus due to head injuries which become epithelialised, then sink into the skull and remain indrawn. In such cases the intracranial pressure is obviously not increased and the possibility of abscess formation can almost be excluded. These cases of transient fungation may be due to a mild meningeal infection or to any cause

which temporarily obstructs the circulation of the cerebro-spinal fluid.

Abscesses due to Infections of the Face, Scalp & Skull.

This is a heterogeneous group not clearly defined from the preceding one, but consisting of cases in which the infection has spread to the brain by direct extension from overlying parts, as opposed to implantation by a penetrating wound. Two cases (e.g., Case 17) were due to wound infection following protracted operations for the removal of parasagittal meningiomata. One was due to a furuncle in the eyebrow (Case 18), one to a carbuncle of the scalp, one to a boil on the cheek leading to cavernous sinus thrombosis (Case 20), and one to osteitis of the skull following a dental extraction (Case 19). In four of the cases there was clear evidence of osteitis of the skull and spread of infection to the brain was presumably by this route. In two cases there was no evidence of bony infection, and in these it was assumed that the extension had been by way of infected venous channels: such a process was demonstrated in Case 20.

That a superficial infection can spread to the brain through an intact skull is well known. The first brain abscess which I encountered followed a trivial laceration of the outer end of the right eyebrow which became infected and suppurated for two weeks before healing took

place. There was no clinical or radiological evidence of a bony lesion. This patient died suddenly about four months later, and at the autopsy there was an abscess in the anterior part of the right frontal lobe. The skull overlying it was intact and healthy. In such a case the presumption is that the infection traverses the skull by way of venous channels. But whatever the mechanism the possibility of an intracranial extension from an apparently trivial superficial infection cannot be emphasised too strongly, and it assumes special importance in military practice because of the high incidence of scalp wounds. Such wounds, if treated properly, heal in a few days and leave no sequels; if they suppurate, a brain abscess may result. It is already clear that scalp wounds deserve much more careful attention than they have received in the past, and that the treatment which ensures primary union will prevent many cases of brain abscess.

One case in this group deserves special mention. In Case 20 a brain abscess resulted from a cavernous sinus thrombosis. I have recently seen a similar case not included in this series. Up until the present time this complication has been of little importance because cavernous sinus thrombosis has been almost invariably fatal. Advances in chemotherapy have brought it within the range of curable diseases, however, and Trevor Roper (10) has recently collected twelve cases from the literature

of the past five years. As with disease of the mastoid and frontal sinus, the possibility of curing a cavernous sinus thrombosis and leaving an unresolved brain abscess will have to be considered. In Case 20 the infection had reached the brain by direct spread along venous tributaries of the cavernous sinus: no bony infection could be demonstrated.

It will be understood that the abscesses in this group are in that part of the brain immediately underlying the superficial infection. That is to say that for the most part they are on the supero-lateral surface of the hemisphere and thus readily accessible for treatment; Case 20 provides an exception (Fig.50 p.243) in that the abscess developed in the depths of the temporal lobe, having spread from the cavernous sinus. The results of treatment have been encouraging: taking this and the preceding group together, there were 10 cases of which two ended fatally, and in both of these the diagnosis was not made before death (Case 20 and the gunshot wound described on p. 22). There should have been no special difficulty in dealing with the occipital abscess due to the gunshot wound, but that due to cavernous sinus thrombosis might have been inaccessible owing to its depth in the hemisphere.

III. Pathology of Brain Abscess.

The paths by which infection reaches the brain have been discussed in the preceding sections. It remains to describe briefly the formation of a brain abscess once the infection is established. As with abscesses in other parts of the body, the initial response is a focus of necrosis and liquefaction, with great oedema of the surrounding brain. This lesion will produce focal neurological signs if it happens to be in an eloquent part of the brain and it contributes to the general picture of toxæmia. In the first few days the inflammation is not clearly localised, and this phase is sometimes described as focal purulent encephalitis. By extending into the ventricular or sub-arachnoid systems it may be the direct cause of death from pyocephalus or diffuse purulent meningitis. But as serious as the infection itself is the oedema which causes an increase in intracranial pressure in much the same way that a rapidly-growing tumour does, with the same potentialities of herniation of the cerebrum through the incisura tentorii and of the cerebellum through the foramen magnum. These herniations obstruct the circulation of the cerebrospinal fluid and cause a further increase in pressure. It is this factor which causes the intense headache, the stupor, and the other well-known symptoms of increased intracranial pressure, and it is a common cause of death in cerebral

infections.

If neither infection nor oedema causes death in the early stages, the inflammation becomes localised and the abscess will begin to develop a definite wall or capsule. The histological changes have been demonstrated by Russell, MacFarlan, and Falconer (11) in abscesses produced experimentally in rabbits. They found that at the end of 12 hours the central necrotic area was surrounded by a zone of polymorphonuclear leucocytes. At 24 hours there was a definite core of pus, the surrounding tissues being infiltrated with leucocytes, but the organisms were confined to the pus. By the end of 48 hours the leucocytic defence was aided by the mobilisation of microglial cells which contribute by the ingestion of organisms and the products of necrosis. At the end of 3 days the vascular reaction to the inflammation is apparent in cellular infiltration of the adventitial sheaths of perforating vessels and an increase in the reticulum fibres in the walls of the vessels. By the end of one week, a fine layer of reticulum fibres could be seen between the vessels just beyond the necrotic zone. This development is most marked in that part of the abscess nearest the cortex. By the end of three weeks definite layers in the wall of the abscess could be identified. The central core of pus was surrounded

by a layer of closely-packed foam cells (derived from the microglia) supported by a frame-work of delicate reticulum fibres. A middle layer was composed of small lymphocytes, plasma cells, foam cells, and spindle fibroblasts enmeshed in a circumferentially-arranged reticulum fibre network, strengthened by the presence of fine collagen fibres. A less definite outer zone contained fewer reticulum fibres, sparse inflammatory infiltration with lymphocytes, plasma cells, and reacting microglial cells, and some proliferation of fibrillary astrocytes. From the time the capsule is complete the most important development is **thickening** of the outer part of the middle zone by increase in the collagen and reticulum fibres. As this began in that part of the abscess nearest the surface of the brain, it is last to appear in the deepest part, i.e., that part which is closest to the ventricle, and it may be three or four weeks before the central zone of necrosis is completely invested by a well-defined capsule. An extension from the depths of the abscess may thus rupture into the ventricle, or it may lead to the formation of another younger loculus of the original abscess.

For practical purposes, i.e., with regard to treatment, the formation of a capsule is the most important feature in the development of an abscess. It implies that the

resistance of the host is able to contend with the virulence of the infecting organism, and the initial inflammatory focus which might become a fatal purulent encephalitis is localised to form an abscess. The defences are derived from the leucocytic and vascular responses common to abscesses in other parts of the body, but they are supported by the microglial reaction which is peculiar to the central nervous system. Even though the abscess continues to expand, the passage of time allows the wall to become thicker, and the risks of rupture into the ventricle or subarachnoid space are correspondingly lessened. Moreover, the presence of a wall is of considerable help in whatever method of treatment is employed, as will be seen presently (p.114 - Treatment). In this connection it should be stressed that as the capsule derives the greater part of its strength from the mesoblastic response (reticulum and collagen fibres), it is thickest where it is near the surface of the brain and the rich network of vessels in the cortex and leptomeninges. At a time when the superficial part of the capsule is several millimetres thick, the deepest part may be only a thin, filmy membrane.

As to how long capsule-formation takes in the human subject, information is not abundant: Penfield is quoted by King (12) as saying that although the capsule begins

to form in the first week, it is not thick enough to offer resistance to an exploring cannula until after 2 or 3 weeks. Lebert (13) in 1856 reported records of a thin membrane as early as the 13th day in one case, and on the 22nd and 24th days in two other cases. Grant (14) said that a firm capsule is established between the fourth and sixth weeks. In the present series, of those cases in which the age of the abscess could be assessed accurately (as in Cases 9, 14, 15) the capsule was sufficiently thick at the end of eight weeks to allow the abscess to be dissected from the brain without rupture.

Having become encapsulated, the abscess continues to expand slowly by increased tension within its walls. It may cause a progressive increase in intracranial pressure and, as mentioned above, if the relation to the primary infection is remote in time, the lesion may come to be regarded as a neoplasm. Rarely, spontaneous resolution seems to occur and the thick-walled sterile cavity may be a chance finding at autopsy. Or, as with some cases of gunshot wounds, the abscess may develop extraordinarily slowly over a period of years. In other cases there may be occasional exacerbations of symptoms of a chronic abscess with long periods of well-being in the intervals. These are all uncommon occurrences, however, and once an abscess has formed it usually progresses steadily to produce urgent symptoms endangering life and vision.

This brief description of the development of an abscess might suggest that there is usually a single locus. In fact, in nearly half of the cases of this series there were multiple loculi, even though the main abscess mass was fairly discrete. A shower of infected emboli is the explanation in some cases (Case 1); in others the different thickness of the walls of the loculi suggests that some are "younger" than others, and the presumption is that they are "buds" from the primary focus in the brain, as indicated in the experimental work described above. Another explanation in some cases (Cases 14 and 15) is that the infection has been carried from the primary locus by operative intervention: an exploring needle may transfix the abscess and implant the infection in the surrounding brain before the pus is detected in the primary locus. Thus, the abscesses due to frontal sinus and mastoid infections are often unilocular, if they have not been subjected to attempts at drainage. In some cases the loculi may communicate with one another, forming a sort of honey-comb, but the communication is incomplete, and drainage of one locus cannot be relied upon to drain the whole mass. This conception of the complexity of the abscess cavity is of great importance in treatment, as will be seen presently.

It has been mentioned that the infection may spread

to involve the ventricular and subarachnoid systems at any stage in the development of a brain abscess, and before the days of chemotherapy this was a common cause of death. Even now a frank rupture of the abscess or a gross contamination of these spaces by leakage may be a terminal event which nothing will check. But there are often phases of mild clinical meningitis during the development or treatment of an abscess which are not necessarily very dangerous. Thus, as in Case 10 and 12, there may be a mild febrile reaction, with headache, stiffness of the neck and legs, and a few hundred cells in the cerebrospinal fluid when the infection is traversing the subarachnoid space in entering the brain; or there may be a fairly sharp generalised meningeal reaction when the adhesion of the brain to the dura over an infected bone is taking place; and there may be occasional short bursts of meningitis during treatment for which no cause other than a little leakage can be determined.

IV. Bacteriology of Brain Abscess.

No mention has yet been made of the infecting organism, and indeed it is difficult to assess the importance of the nature of the organism in relation to the pathological changes described above. A wide range of bacteria

(pneumococcus, streptococcus, staphylococcus aureus, bacillus tuberculosis, bacillus fusiformis, etc.) produced abscesses which responded well to treatment and the same organisms were encountered in the fatal cases. It thus seems that there was no specific relation between the type of organism and the mortality. Doubtless some strains are more virulent than others, or the natural defences are less effective against some organisms than others: Russell et al. (11) failed to produce a localised pneumococcal abscess in the rabbit, all cases so infected dying with purulent meningo-encephalitis before abscess formation occurred. They had a high percentage of successes with staphylococcus aureus and anaerobic streptococci, however, and it would seem that the rabbit can cope with these organisms better than with the pneumococcus.

In a recent study of the bacteriology of 47 cases of brain abscess MacFarlan (15) found pure cultures in 33 cases and mixed infections in 14 cases. Of the pure cultures the organism was staphylococcus aureus in 13, strept. pneumoniae (pneumococcus) in 6, strept. pyogenes in 4, Proteus vulgaris, non-haemolytic streptococcus, and B. fusiformis in 2 each; anaerobic streptococcus, strept. viridans, micrococcus tetragenes, haemophilus influenzae in 1 each. In the mixed infections, the commonest organisms were fusiform bacilli (8 cases) and anaerobic

streptococci (9 cases). That earlier reports showed a predominance of streptococci over staphylococci may mean that the chemotherapeutic agents available in the last few years have been more effective in dealing with streptococcal infections which might have given rise to abscess of the brain.

There is no evidence that some organisms favour capsule-formation more than others. Whether or not an abscess develops a capsule depends on the relation between the virulence of the organism and the resistance of the host, and how thick the capsule becomes is a matter of the length of time which elapses before the abscess demands treatment. It has been stated by Alpers (16) and Grant (17) that anaerobic and gram-negative organisms actively inhibit capsule-formation, but both types of organism were encountered in several cases in this series which had well-formed capsules, and Russell et al (11) were not able to demonstrate any such inhibitory effect in experimentally-produced abscesses containing these organisms.

At present it seems that of more importance than the nature of the organism is its sensitivity to modern chemotherapeutic agents. The effect of these agents on the brain abscess itself is problematical, but there is no doubt about their effect on the primary focus of the

disease from which the brain abscess originates, and on the meningitis which may occur during the treatment of an abscess.

V. Symptoms and Signs of Brain Abscess.

The clinical picture of a patient suffering from abscess of the brain is well known, but as with so many other well-known clinical pictures it is usually the too-late stage of the disease which is described. The clinical state is a combination of toxaemia, increased intracranial pressure, and signs of a focal lesion of the brain. The toxaemia may be derived partly from the abscess itself, or it may be due to the primary disease responsible for the abscess, e.g., bronchiectasis. The headache, visual failure, vomiting, somnolence and coma are symptoms of increased intracranial pressure due partly to the size of the abscess and partly to the surrounding oedema. The focal signs - such as aphasia, defects in the fields of vision, hemiplegia - are due to the local effects of the abscess which may destroy the part of the brain subserving these functions, in which case there will be a permanent loss of function even though the abscess is treated successfully. More commonly the abscess exerts local pressure on neurones

and fibre tracts, and the focal neurological abnormalities will clear up when the abscess is treated. There may be more diffuse and less profound effects at a distance from the lesion due to extensive oedema or indirect pressure disturbances: thus an abscess at the frontal pole may produce slight changes in motor power and sensation even though it is at a considerable distance from the relevant cortical areas and fibre tracts.

Treatment has the greatest chance of success if the case is brought under skilled observation before these gross signs are manifest. Early diagnosis may demand many of the refinements of modern medicine, but more often than not the diagnosis is missed because of its not being considered. The possibility of **intracranial** extension should be borne in mind when dealing with any infection, or potentially infected wound of the head. This applies to scalp wounds from the moment of their infliction until healing by primary union has occurred; and it is even more obvious in every case of mastoid and frontal sinus infection. It has been shown that the relation of cerebral symptoms to a preceding infection in some other part of the body may be obscured by the passage of time or by the apparently trivial nature of the primary infection, and in some of these cases the diagnosis may not be established until the lesion is

exposed for treatment. In this series the diagnosis was missed altogether in 3 cases: one due to cavernous sinus thrombosis (Case 20), one to gunshot wound (p. 22), and one case of multiple cerebral and cerebellar abscesses due to staphylococcal bacteraemia. The first and third cases were examples of misinterpretation of the evidence, as in both an abscess was suspected. In the case of the gunshot wound also an abscess was suspected, but special investigations were thought to exclude it three months after the injury. This case should have been kept under regular observation.

Although there were only 3 cases in which the diagnosis was not established until after death, there were 6 others in which the nature of the lesion was in doubt until it was exposed at operation. Four of these were metastatic abscesses from apparently trivial superficial infections; one was a fluid tuberculous abscess in the cerebellum; and one was a chronic temporal abscess in a child of eight years whose mother did not remember an aural infection three years earlier until after the operation, despite direct enquiry. Thus in 18 per cent. of the cases in this series the pathological diagnosis presented special difficulties. This figure is an index of the problem in a neurosurgical clinic where special diagnostic facilities are available and where the possibility of an abscess usually receives careful consideration.

A full description of the semeiology of brain abscess would demand an account of almost every symptom and sign of intracranial disease, and that is neither necessary nor desirable. There are, however, some general observations which deserve special mention and some characteristic features of abscesses in particular sites which are worth recounting. It should be said at the outset that just as almost any symptom or sign of intracranial disease can be encountered in a case of brain abscess, so almost any symptom or sign which might be expected or looked upon as essential for the diagnosis can be absent. This accounts for some of the difficulties of diagnosis, and even when all the facts are known at operation or autopsy, it may be impossible to explain some of the clinical features.

The onset of the infection may be sudden and dramatic or it may be so insidious that the patient and his relatives cannot assign even an approximate date to it. The sudden onset may betoken the lodgement of infected emboli, as in Cases 1 and 2, and the clinical manifestation may be either an epileptic fit with focal characters or the abrupt onset of a hemiplegia. Such episodes, of course, rarely escape notice. Less common, but no less eloquent, are transient attacks of unorganised visual hallucinations (Case 7), aphasia, and disorders of spatial

orientation (Case 5). These disturbances of cortical function differ from similar disturbances due to other causes (e.g., cicatrix, neoplasm, etc.) in that recovery is apt to be less complete and the residual defect becomes progressively more marked even when the immediate effect of the attack has worn off. Thus in Case 7 the visual hallucinations in the left homonymous field were immediately followed by a permanent defect in the field which had become a complete homonymous hemianopia in five weeks. An abscess in the Rolandic region may start like a bolt from the blue with a Jacksonian attack in the opposite arm; the attack is immediately followed by complete paralysis of the arm, but this largely recovers within a few minutes or hours. Recovery is not quite complete, however, and each succeeding attack leaves more residual weakness; or even if there are no more attacks the weakness progresses rather more rapidly than is the case with most neoplasms.

In some cases of metastatic abscess the sudden onset of headache may be the first symptom. Not uncommonly the headache may be accompanied by pain in, and stiffness of, the neck, nausea and vomiting, and photophobia. These characters are reminiscent of meningitis, and although a case rarely comes under observation at this stage it is probable that the headache is due to a sharp

meningeal reaction provoked by the lodgement of infection near the surface of the brain. Increased intracranial pressure is unlikely to be the explanation at the onset, as the processes contributing to an increase of pressure will take at least some hours to develop. As time goes on, however, the meningeal signs may be replaced by headache of the type commonly associated with increased pressure, and this is due to expansion of the abscess and surrounding oedema.

In other cases of metastatic abscess the onset may be not remarkable. It may occur during a period of ill-health, as when a person is experiencing a siege of boils, and there may be nothing to call the attention of the patient or his doctor to the beginning of the intracranial infection. In these cases headache is usually the first symptom: it begins gradually, but becomes more persistent and severe, and is in no way distinguishable from the headache due to other causes of increased intracranial pressure, e.g., neoplasm. Indeed, in 6 of the cases in this series (Table I, Group I C.) the lesion was regarded as a neoplasm until it was exposed at operation.

In the case of mastoid infections it may be impossible to recognise the onset of the intracranial extension. In one case in this series a thick-walled temporal abscess produced no symptoms until 3 weeks before operation and from its appearance it must have been present for many

months, if not several years. More commonly there is something during the course of the mastoiditis to suggest that something else is happening, and this may occur before or after the mastoid has been operated on. In Case 8 it was the onset of headache after spontaneous resolution of a middle-ear infection; in Case 9 it was the persistence of pain in the ear after the drum had ruptured, and later the sudden onset of nausea, vomiting and headache when the symptoms of the aural infection had disappeared. In Case 10 it was stupor and incontinence after a mastoid operation, without much headache; in Case 11 much more severe and protracted pain than is common with chronic mastoiditis. In another case not described at length it was the persistence of headache and of a very profuse purulent discharge from the ear after the mastoid wound had healed soundly. In other cases it is the appearance of focal neurological abnormalities which will be described presently.

Epilepsy is not a common occurrence in cases of abscess due to mastoiditis. It would not be expected in the cerebellar abscesses, but in none of the 11 temporal lobe abscesses did it occur. This is in marked contrast to the frontal abscesses due to sinusitis in which an epileptic fit occurred during the incubation of the abscess in 4 out of 6 cases. I have seen one case

of temporal abscess due to mastoiditis not included in this series in which there was a focal motor attack involving the opposite side of the face and arm, but this must be regarded as uncommon.

If the infection spreads from the frontal sinus, a characteristic feature is that the headache is rather more severe and prolonged than is common with acute frontal sinusitis. It is definitely a headache, as opposed to the local pain of sinusitis, and it may be accompanied by drowsiness, nausea and vomiting. It may be difficult or impossible to dissociate these symptoms from those commonly met with in acute sinusitis, and unless there is something more dramatic, such as an epileptic fit, the onset of an intracranial extension may not be recognised. It has been said **above** that an epileptic attack is a very common feature of developing frontal abscesses, and such an incident is so significant that the case should not be allowed to escape from observation until the possibility of an abscess has been excluded, even though other symptoms and signs have cleared up. It has also been shown (p. 17) that in every case of frontal abscess due to sinusitis in this series there was oedema, redness and local tenderness of the forehead during the acute stage of the sinus infection. A brain abscess does not always result when these signs

are manifest: they probably mean nothing more than a focal osteitis which may resolve. But the relation of these signs to subsequent abscess formation is so striking that the case will demand continued observation. There are, of course, some cases of frontal sinusitis (as of mastoiditis) which do not come under observation in the acute stage or which resolve spontaneously; if a brain abscess develops in these cases it may present the symptoms and signs of any other expanding lesion of the brain.

Most ear, nose and throat surgeons have their own personal standards for the normal course of mastoiditis or frontal sinusitis and for post-operative convalescence. It cannot be emphasised too strongly that any deviation from this course should raise the question of an intracranial extension, and diagnostic steps taken to exclude such complications before the case is dismissed. The investigations would include a clinical examination of the nervous system - and especially an inspection of the optic fundi and assessment of the visual fields - lumbar puncture for measuring the spinal fluid pressure and for analysis of the fluid, and in some cases encephalography or ventriculography. In a large proportion of cases these investigations will reveal no abnormality, but only by pursuing them will it be possible to avoid missing the diagnosis in a few cases.

In the case of penetrating wounds abscess formation should be suspected if the wound fails to heal by primary union or heals and later suppurates. Superficial suppuration may, of course, be confined to the scalp or skull, but if it persists it can ultimately cause a brain abscess. The early stages of abscess formation may be missed because of the immediate effects of the injury: a person with a severe compound fracture may be unconscious for several hours or days, stuporose for several more days and unable to co-operate in an examination. When he does recover sufficiently to allow a detailed examination the neurological abnormalities may have been present from the time of the injury or they may have been progressive. It is thus of the utmost importance to get base-line readings as early as possible: neurological effects of an injury are maximal immediately after the injury, and any evidence of progression should raise the suspicion of abscess formation. The appearance of a cerebral hernia or fungus will in most cases point to an intracranial abscess, as in an uninfected case without increased pressure a depression occurs at the site of the bony defect within ten to fourteen days of the injury (Fig. 10).

With infections of the face, scalp and skull the symptoms of intracranial extension are varied. In the

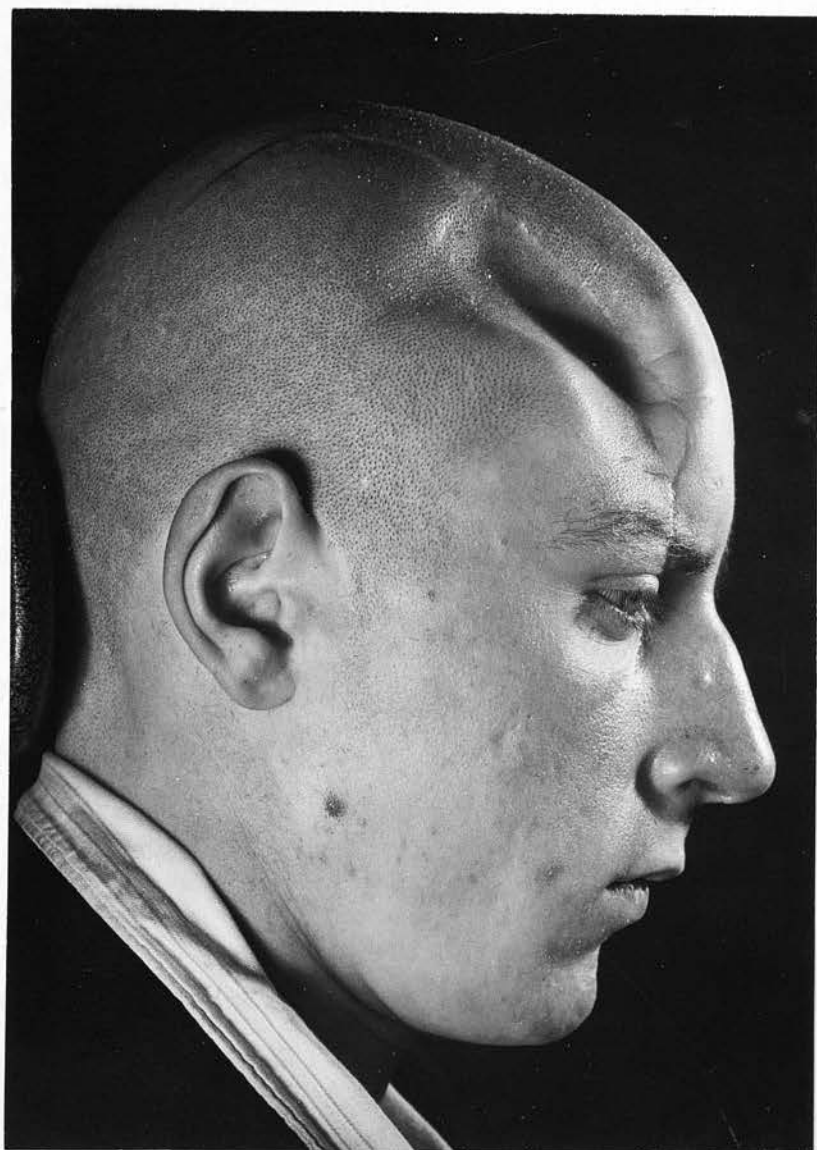


Fig. 10. Showing the depression which occurs after a compound fracture of the skull, with loss of the fragments. The wound healed by primary union, and the depression was apparent about fourteen days after the injury. It was subsequently repaired by a rib graft.

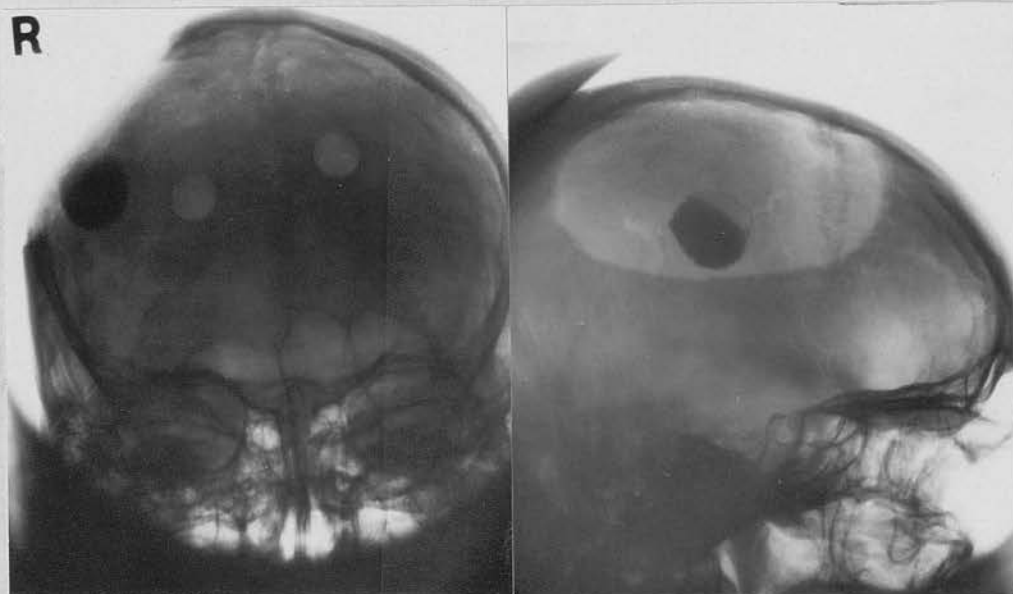
two cases of infection of an operation site there was suppuration of the scalp wound and later evidence of osteitis of the bone flap. In Case 17 there were no intracranial symptoms until ten weeks after the primary operation and one week after the operation for the removal of the infected bone flap. Then it was headache, malaise, a fairly sharp meningeal reaction and the gradual onset of a hemiparesis. In Case 18 the first symptom was the onset of severe paroxysms of headache five weeks after an abscess of the right eyebrow had been drained. In one case not reported at length the onset of the intracranial infection was probably signified by a severe mental disturbance necessitating certification.

This was a young man who developed a carbuncle in the right parietal region. It was incised, and a few days later he was very ill with a staphylococcal septicaemia. This was treated successfully with penicillin, but during convalescence, when it was thought that the infection had been mastered, he rapidly became demented, presenting the picture of acute mania. The scalp wound had healed, and there was no constitutional evidence of unresolved infection. Neither were there any neurological abnormalities until a fortnight later, some six weeks after the carbuncle had appeared, when he complained for the first time of some local pain and tenderness of the

skull just anterior to the scar of the carbuncle. Within a day or two a fluctuant swelling appeared at this site, and X-rays showed evidence of osteitis of the skull in the vicinity. As this swelling occurred, he developed a rapidly-progressive weakness of the left side of the face, left arm and leg. An operation at this stage revealed a sub-galeal abscess (to account for the fluctuant swelling in the scalp), diffuse osteitis of the parietal bone (which was largely removed, Fig. 11), a large extradural abscess beneath the parietal bone, and a mass of extradural granulation tissue over the lower part of the exposed dura. The wound healed soundly and there was immediate and striking improvement in his mental state and in the hemiplegia. By the end of three weeks he appeared to be normal mentally and there was only a slight residual weakness and sensory loss of a cortical type in the left upper limb. He then began to deteriorate as regards both the mental and the neurological condition. Within a few days he was quite unmanageable: talking incessantly, writing meaningless letters to people with whom he had no need to correspond, throwing objects about his room, breaking doors, windows, etc. During this phase he was very cheerful and affable and admitted to no symptoms. Two or three days later, however, the hemiplegia recurred, the spinal fluid pressure was elevated, and the scalp was more tense over the craniectomy. A ventriculogram

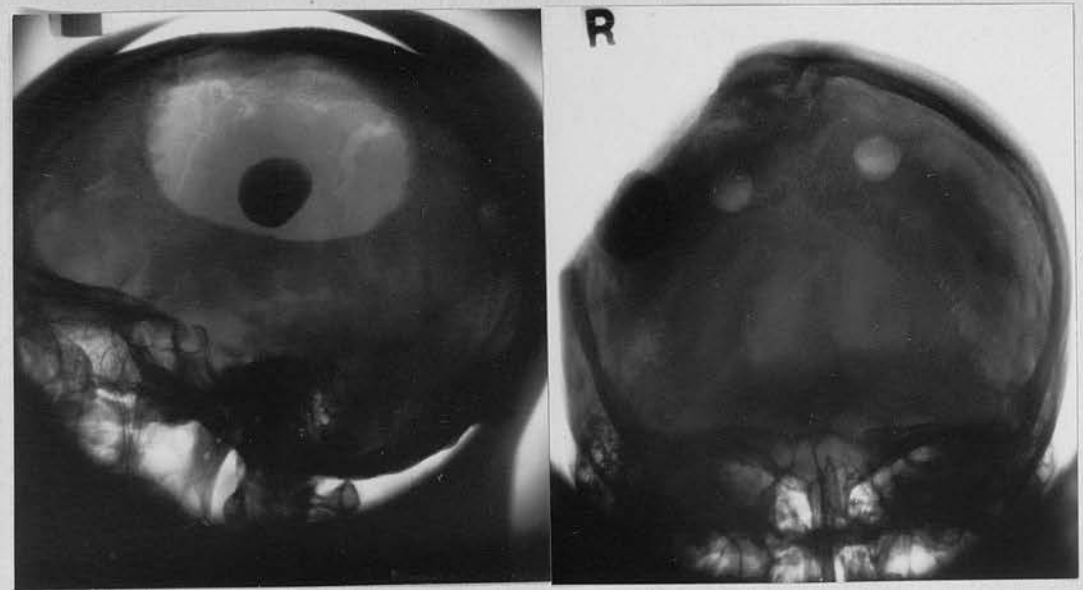


24th. December, 1943.

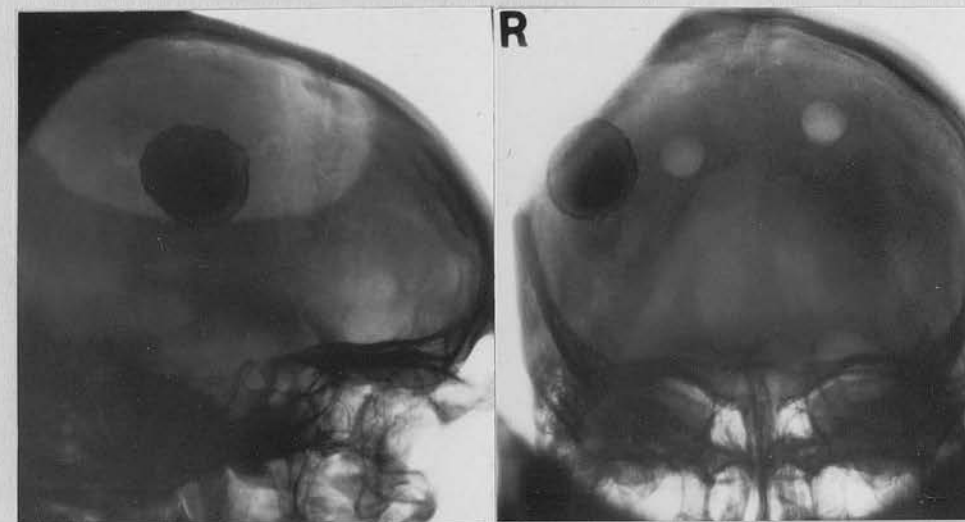


25th. January, 1944.

Fig. 11. Right Rolandic abscess due to carbuncle of scalp and osteitis of skull. The plate taken on 24th. Dec., 1943, shows an area of erosion in the right parietal region. Those on 19th. January show the extent of the removal of the infected bone, and thorotrast in the cerebral abscess. Those on the 25th. January and 16th. February show a progressive increase in the size of the abscess, and its tendency to be pushed into the decompression area.



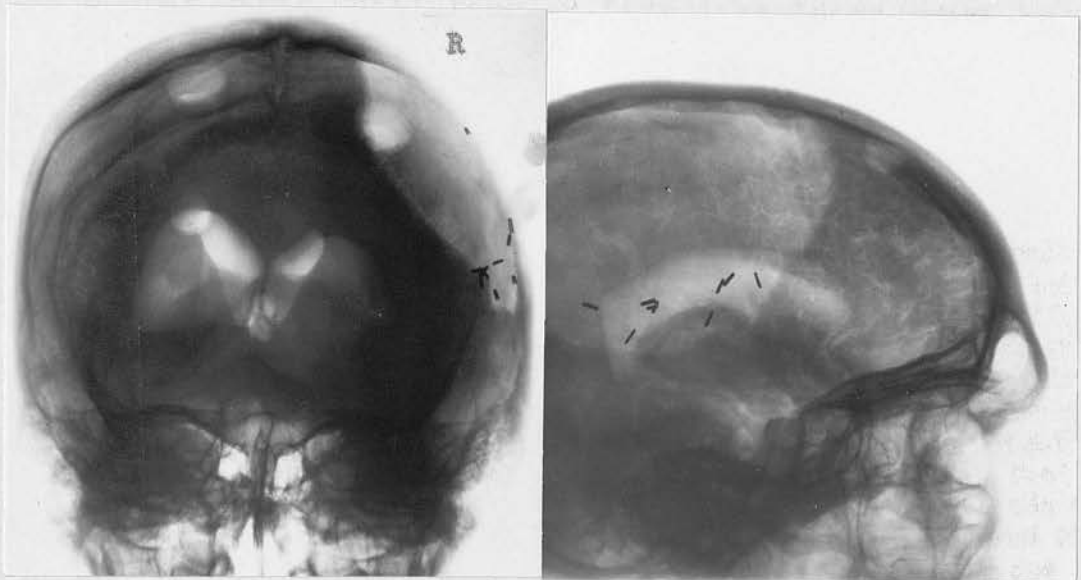
19th. January, 1944.



16th. February, 1944.



10th. March, 1944.



12th. April, 1944.

Fig. 12. Continuing Fig. 11, page 50 a, the plate taken on 10th. March shows the size of the abscess immediately prior to operation on that day. The ventriculogram was taken four weeks after the abscess was extirpated from the right hemisphere, and shows the slight relative dilatation of the conralateral (left) ventricle, the absence of deformity of both ventricles, and the absence of lateral displacement. Prior to operation the right ventricle was collapsed and the left ventricle was markedly displaced to the left side.

indicated total collapse of the right ventricle and marked displacement of the left ventricle to the left side. A sharp needle was introduced through the scalp flap over the site of the extradural granulation seen when the bone was removed. An abscess containing 25 c.c. of thick yellow pus (which yielded staphylococcus aureus, as did the extradural abscess and the infected bone removed at the first operation) was evacuated by aspiration and 2.c.c. of thoro-trast instilled (Fig. 12). Again there was immediate improvement in the hemiplegia, but there was no improvement in his mental state until four or five days later, when he rapidly became quite normal and remained so. The site of the craniectomy continued to bulge slightly, the spinal fluid pressure remained at about 250 mm., and although the protein content was normal in repeated examinations there were always 6-10 cells per c.mm. Of neurological abnormalities there was some residual weakness of the left side of the face and of the left upper limb, with a cortical type of sensory disturbance in the hand. There was no papilloedema and at no time any headache, but the continuing signs indicated that the abscess needed further **treatment**, so the flap was re-elevated ten weeks after the onset of the intracranial infection and the abscess dissected out in toto. It had a firm capsule and the dissection presented no special difficulties. The abscess was



situated just beneath the cortex in the lower end of the Rolandic region, if anything slightly posterior to the central sulcus. The excision thus entailed the sacrifice of some valuable cortex, and he was left with a considerable weakness of the left upper limb, and to a less extent of the left side of the face and the left lower limb.

In some cases of abscesses due to septicaemia it may be difficult to dissociate symptoms of abscess formation from cerebral symptoms due simply to toxæmia, e.g., delirium, stupor, incontinence, etc. The same may be said of abscesses following cavernous sinus thrombosis (Case 20) and of other diffuse infections of the bone and soft tissues of the face and head. But in all the persistence of headache and the appearance of neurological signs should raise suspicion.

Once the infection has become established and the abscess begins to grow, headache is the most constant and characteristic symptom. It is due to increased intracranial pressure and may be generalised or referred particularly to the front or back of the head and neck. We have not found that reference to one or other side of the head has any constant value in localisation, but some patients with temporal lobe abscesses due to mastoid disease are definite in saying that the headache is largely confined to the side of the lesion. In some cases, as in Case 11, this lateralisation may mean the presence of an

extradural abscess. The headache may be constant, but it is usually of variable intensity, being worse on waking in the morning, aggravated by stooping, straining or coughing; not much affected by ordinary analgesics, such as aspirin, but somewhat relieved by dehydrating measures, e.g., the rectal administration of magnesium sulphate; it is frequently accompanied by vomiting, and the act of vomiting aggravates the headache. These are all features of headache due to increased **intracranial** pressure, whether due to abscess, neoplasm, or other causes. In some cases the headache may occur in short paroxysms, with complete freedom in the intervals; this was particularly marked in Case 18, a frontal abscess.

In three cases of cerebellar abscess we have noticed that the headache is relieved by bending the head forward, and these patients were often seen with their heads lifted from the pillow in an uncomfortable-looking attitude, resenting any attempt to extend the head. In cases of cerebellar tumour, on the other hand, or of pain in the neck due to increased pressure from whatever cause, the patient often prefers to have his head slightly extended and resents any attempt at flexion. In some cases of acute rise in pressure the head may be sharply extended and the back arched in an opisthotonic attitude: this occurred in a case of temporal lobe abscess in a child

of eight years shortly after admission to hospital. Although her attitude was very alarming she was quite conscious and co-operative, said that she had very little headache and that in fact it was easier than before the opisthotonus had occurred. There was none of the disturbance of pulse rate, respiration and blood pressure commonly seen in acute increases in pressure. This lack of distress has been observed in similar cases of opisthotonus due to tumours in the posterior fossa, but whatever the cause it is an indication for immediate treatment, as sudden death may occur at any time.

An important feature of the headache associated with brain abscess is that it may clear up without treatment. Indeed in one case (Case 10) of temporal lobe abscess there was little, if any, headache throughout the illness, stupor being the predominant feature. The patient was an elderly woman, and it has **long been** known that brain tumours can attain a considerable size in old people before causing increased intracranial pressure. The explanation commonly offered is that the total subarachnoid space is more capacious in old age and affords more room for an expanding lesion to take up before the circulation of the cerebrospinal fluid is obstructed and the intracranial pressure rises. But this explanation does not apply to the cases in which headache is a prominent

feature in the early stages of abscess formation and then clears up as the abscess becomes more chronic. There were eight such cases in this series, all in abscesses of the cerebral hemispheres. In one of them, a child of five years, the explanation was probably that the skull enlarged by separation of the sutures, thus affording a kind of natural decompression effect as frequently happens in cases of slowly-growing intracranial tumours in childhood. In the remainder the explanation is not clear: despite the freedom from headache, papilloedema developed, or if already present it progressed, in one case to be followed by complete blindness due to secondary optic atrophy. In all of these cases the spinal fluid pressure was persistently elevated to 250 mm. or more; and in some of them the effects of long-standing increased pressure were apparent in the X-rays of the skull which showed convolitional thinning of the vault and erosion of the sella turcica.

The disappearance of headache is, of course, neither characteristic of nor peculiar to brain abscesses. It has been observed in many cases of intracranial tumour of all sorts and locations, and the explanation is equally obscure. A common story is that the headache ceases when blindness occurs from secondary optic atrophy, although there is nothing else to suggest any appreciable

reduction of intracranial pressure. It is not that these people do not feel headache (there are many such people) or do not know how to differentiate a headache from a pain in the head or are so advanced in stupor or dementia that their sensibility to pain is dulled. They have all experienced headache, know what it is, and may be quite alert and intelligent when they become free from it. Indeed, once the headache clears up there may be good general health and nothing on clinical examination to show that there is an active intracranial lesion except progressive papilloedema. Whatever the explanation, the inference is obvious: no case suspected of having or known to have had a brain abscess should be allowed to escape observation until it is certain that the intracranial pressure is normal and remains so.

In some cases of chronic abscess the headache may occur in attacks lasting two or three days at a time, with long periods of freedom for weeks or months between the attacks. In case 13, a chronic cerebellar abscess, these attacks were regarded as migrainous phenomena, as no abnormal neurological signs were noted and there was good health in between the attacks.

Despite these occasional vagaries, headache remains the most constant feature of brain abscess. But it is such a common symptom of so many and so diverse affections

that objective evidence of increased intracranial pressure may be needed for diagnosis. Such evidence is afforded by lumbar puncture and measurement of the pressure in the spinal theca. The risks of this procedure in cases of increased intracranial pressure are well known, but it is reasonably safe in circumstances which allow a crisis to be dealt with by operation if necessary. Apart from the information gained by manometry, the examination of the spinal fluid is of so much importance in diagnosis (vide infra, pp.61-66) that lumbar puncture is a part of the routine investigation in this clinic.

Examination of the optic fundi also yields valuable information in some cases, but not in all: indeed, in 20 of the cases in this series the fundi were normal throughout the illness. Whether or not papilloedema develops seems to depend largely on the duration of the increased intracranial pressure, and in those cases which prove fatal within a few days or in which the abscess is treated at an early stage no marked changes in the fundi will be expected. Of the cases in which the age of the abscess could be determined accurately there was one (Case 6) in which there was definite papilloedema at the end of four weeks; and some which came under observation in the fifth and sixth weeks had well-marked swelling of the optic discs. On the other hand, there were some cases of

chronic abscess with a long history of headache in which the fundi were normal: this was an interesting feature of Case 13, in which a cerebellar abscess had been present for long enough to produce a moderate internal hydrocephalus (Fig.40 p.212) and yet there was no papilloedema. Examination of the optic fundi thus may yield valuable positive evidence of increased intracranial pressure, but the absence of papilloedema should not be taken as unequivocal evidence against the presence of an expanding lesion, whether it be an abscess or a neoplasm. It has already been mentioned that papilloedema may develop insidiously in chronic abscesses, even though the headache and other symptoms of increased intracranial pressure have cleared up, and this means that a careful watch should be kept on the fundi and the visual acuity to prevent permanent loss of vision from secondary optic atrophy.

Of the other general symptoms of abscess formation stupor is one of the most important. It may be a feature of toxæmia, as in cases of septicaemia, empyema thoracis, endocarditis, etc., but more commonly it is due to increased intracranial pressure. If the latter, it is a late sign and may be the immediate precursor of coma and death. But there are some cases in which it seems to be not so much a factor of increased pressure as of local oedema: thus in Case 9, a right temporal abscess due to mastoiditis,

there was coma without any objective evidence of increased pressure - the spinal fluid pressure was normal, as was the intraventricular pressure, and the dura was quite slack when a bone flap was elevated to expose the abscess. When the brain was incised it was found to be very soft and oedematous, and during the dissection fluid could be squeezed from the white matter by pressure with pledgets of dry cotton wool, so that they immediately became waterlogged. In Case 10, another temporal abscess, stupor was the predominant feature, to the exclusion of headache, papilloedema, or increase in the spinal fluid pressure. X

Associated with stupor there is often incontinence of urine and faeces, difficulty in swallowing, and respiratory depression. These symptoms add to the problem of nursing, and unless they are properly dealt with they may actively contribute to deterioration. Thus a stuporose patient who is lying immobile in a bed frequently soiled by urine and faeces is very apt to develop bed-sores. Slowness in chewing and swallowing may lead to inanition in chronic cases, and this adds to the profound general wasting which may occur. Inanition during the period of stupor is often followed by a voracious appetite during recovery, and the "abscess appetite" is a feature well known to the nursing staff.

Of perhaps more importance is the effect of increased

intracranial pressure on a respiratory infection. If a patient with bronchiectasis, lung abscess, or chronic empyema develops a brain abscess, the onset of stupor means that coughing is repressed and the purulent secretions and exudate are not evacuated from the lungs. Thus these patients may die with a fulminating chest infection even before the brain abscess has time to develop to any appreciable size.

The general signs of inflammation are usually of very little help in diagnosis. It is often stated in text-books that the temperature is sub-normal in cases of brain abscess, but we have not found that to be the case: the temperature is usually normal, showing an occasional elevation of one or two degrees. Sharp rises may be associated with phases of meningitis or with some development in the primary focus, such as a flare-up of chronic mastoiditis or a patch of pneumonia in a case of chronic bronchiectasis. The temperature may be considerably influenced by chemotherapy given for the primary condition, and in exchange for the great value of this advance in treatment we have had to sacrifice the information which the temperature often gave about the reaction of the body to infection. So with leucocytosis and the sedimentation rate of red blood cells: these investigations usually reflect the reaction to the primary focus, but are not significantly or constantly altered by the brain abscess alone. As

with the temperature, there may be changes during phases of acute meningitis or in exacerbations of quiescent distant infections during which the cerebral infection begins, and they may draw attention to a primary source of inflammation which might otherwise have been missed.

The pulse rate depends on both the reaction to infection and increased intracranial pressure. During a phase of activity of the primary focus it will be raised, along with the temperature, but if there is no such general reaction the brain abscess by itself will produce no striking alteration until the intracranial pressure is raised. Then the pulse becomes slower and the blood pressure rises slightly. The slow pulse goes with the headache and stupor, and it increases in rate if measures are taken to relieve the intracranial pressure. It becomes very rapid only in the terminal stages of coma, or of a fulminating general infection.

The most reliable objective evidence of a brain abscess is found in the examination of the cerebrospinal fluid. Lumbar puncture was not done in 2 cases in this series; of the remaining 48 there were only 2 in which the cerebrospinal fluid was quite normal, and both of these were chronic abscesses. Thus in over 90 per cent. of cases examination of the fluid was of positive value in diagnosis. The significant change is a slight

increase in the protein and cell content. In the Pathological Department of the Radcliffe Infirmary, where most of the examinations were made, the upper limit of normal is taken as 45 mgm. of protein per hundred c.c. and 2 or 3 cells per c.mm. In 50 per cent. of the cases the protein content was between 50 mgm. and 100 mgm. per cent.; in 35 per cent. it was less than 50 mgm., and in 15 per cent. more than 100 mgm. The low values are more often met with in cases of chronic abscess in which the protein content may be quite normal, but in all except 2 of these cases there was a slight increase in the cell content. The high protein contents were found in cases of acute fulminating abscesses, and in the cases with sharp meningeal reactions, although in the latter the protein content was raised out of proportion to the cell count, suggesting that the infection was not confined to the leptomeninges - i.e., that the fluid was not simply that of a primary meningitis. Thus one case of multiple bronchogenic abscesses showed 110 mgm. protein and 475 cells during a phase of pronounced clinical meningitis. Another case of multiple cerebral and cerebellar abscesses with marked meningeal signs had 240 mgm. protein and 37 cells.

The cellular reaction is an even more delicate index of intracranial infection than the increased protein

content. In 90 per cent. of the cases in which the fluid was abnormal the cell count was raised, and in a third of these the protein content was normal. The pleocytosis is usually a slight one, however: in 70 per cent. of cases the cell count ranged between 5 and 50 per c.mm. Higher counts were met with in some cases of acute abscesses and in the phases of clinical meningitis which may occur during the incubation or treatment of an abscess. Of the cases in which the cell count was normal the great majority showed a significant increase in the protein content, just as in the cases with a normal protein the majority showed an increase in the cell count. During the acute stage of abscess formation the polymorphonuclear leucocytes account for the greater part of the increase, as in the acute meningeal crises; but as the abscess becomes more chronic lymphocytes predominate, and in the late stages of encapsulation all of the cells may be lymphocytes.

Although these changes in the cerebrospinal fluid are so constant, they are by no means pathognomic of brain abscess. Increase in the protein content is a very common finding in cases of intracranial neoplasms of all sorts, and the cell count may be raised with or without an increase in the protein content. Some cases of degenerating and necrotic gliomata situated near the

subarachnoid space or the ventricular system may show a marked pleocytosis, contributed to by polymorphonuclear leucocytes as well as lymphocytes, and in these cases the short history of a progressive intracranial lesion may make the differentiation from an abscess difficult or impossible. Furthermore, it has been shown that in some cases of brain abscess, repeated examinations of the spinal fluid are normal. Such cases form a small but important minority, as they are usually chronic abscesses which will respond to treatment. It should be emphasised, then, that a normal spinal fluid does not exclude the possibility of a brain abscess.

Analysis of other constituents of the spinal fluid show less constant changes and are of less value. The sugar content may be reduced or absent, especially in the phases of meningitis. The chloride content may fall to the lower limit of normal, but rarely lower than that unless there is protracted vomiting or some other general cause for chloride depletion. It should be mentioned, however, that in one case in this series a persistently low chloride content led to the mistaken diagnosis of tuberculous meningitis. This was a case of multiple cerebral and cerebellar abscesses following staphylococcal septicaemia. The chloride content in repeated analyses dropped from 670 mgm. to a low of 590 mgm. The protein content was usually between 100 - 200 mgm., and the cell

count varied between 37 and 953 per c.mm. The low cell counts were essentially lymphocytic, while the higher ones were largely polymorphic. The case presented many clinical problems, and the chloride readings were lower than anything we had encountered in pyogenic infections of the brain or meninges. As no general cause could be found, it was thought that the low chloride content probably pointed to a tuberculous infection, although the tubercle bacillus was never found. The error was demonstrated at the **autopsy**: there were multiple staphylococcal abscesses, with no gross or microscopic evidence of tuberculosis, and indeed no naked-eye evidence of leptomeningitis. The cause of the diminished chloride content was never established.

Not only does examination of the cerebrospinal fluid help in diagnosis, but it affords some index of the progress of treatment and it is an important part of the test of cure (v.i., p.152). Thus a diminution in the cell count may point to the mastery of a meningeal infection, just as a sharp increase may mean an acute meningitis which, if not checked, might prove fatal even though the brain abscess is progressing favourably. A relative increase in lymphocytes usually means that the infection is becoming chronic, and, as stated above, when all of the cells are lymphocytes the abscess is

usually entirely encapsulated. Lowering of the protein content too is a favourable sign, as it tends to occur during encapsulation, and in all of the cases in this series in which the protein content was normal the abscess was in a fairly chronic stage.

The cerebrospinal fluid should return to normal when the abscess has been successfully treated. In this connection, it should be said again that unless the abscess has been extirpated a normal spinal fluid should not be accepted as a test of cure by itself: the normal fluid may simply bespeak quiescence of the infection and the fluid may again be abnormal two or three days later. On the other hand, it cannot be emphasised too strongly that a persistently abnormal spinal fluid in a case of brain abscess under treatment means that the abscess will demand further treatment even though there may be no other signs or symptoms to indicate it (see Test of Cure, p. 153).

Micro-organisms are not found in the fluid unless there is meningitis as well as the brain abscess. It has been shown above that the organisms are confined within the abscess in the early stages of its development and their presence in the spinal fluid means an extension of the infection to the subarachnoid space, and, indeed, that the infection is widely disseminated. Before the

days of chemotherapy this was a common terminal event, but now few cases of meningitis are beyond the range of successful treatment. The outcome depends largely on the sensitivity of the organism to the chemotherapeutic substances at present available.

Radiology in Diagnosis. Ordinary X-rays of the skull are an important part of the routine investigation, but in many cases they yield no information of positive value. They may show evidence of infection of the mastoid or frontal sinus, but there is nothing in these appearances characteristic of an intracranial extension of the infection. Osteitis of the vault of the skull produces definite X-ray changes after three or four weeks, and this may be of value in the diagnosis and localisation of intracranial symptoms and signs, as in the case reported on p. 49. Lateral displacement of a calcified pineal may point to an expanding lesion in one or other cerebral hemisphere, but the lesion may be an abscess, a neoplasm, or some other space-occupying process. The common signs of increased intracranial pressure (convolutional thinning of the vault, and erosion of the dorsum sellae) take several weeks to become apparent, and we have not seen separation of the sutures in childhood in less than 6 weeks from the onset of the infection. In short, there are likely to be no

significant abnormalities in the early stages of abscess formation, and the changes which occur later as the abscess becomes chronic are those due to increased intracranial pressure.

In rare cases ordinary X-rays may give direct evidence of the presence of an abscess. This depends on (1) the presence of calcification in the wall of a very chronic abscess; or (2) the presence of gas in the abscess cavity. We have not encountered either of these findings in this series, and it is perhaps surprising because it is not very unusual for bubbles of gas to escape with the pus aspirated from an abscess. These bubbles may be too small and too sparse to cast a shadow, but the possibility should be borne in mind in interpreting the skiagrams. Calcification sufficient to show in X-rays is much more apt to be due to a calcified glioma than an abscess: in one case of increased intracranial pressure without localising signs the skiagrams showed an even ellipse of calcification (Fig. 13) which we thought looked like a calcified frontal abscess should look, but at operation it proved to be an oligodendroglioma. In other cases regular circles of calcification may be seen in intracranial aneurysms.

Although the scope of straight radiography is somewhat limited in cases of brain abscess in which the skull is intact, the position is quite different in dealing

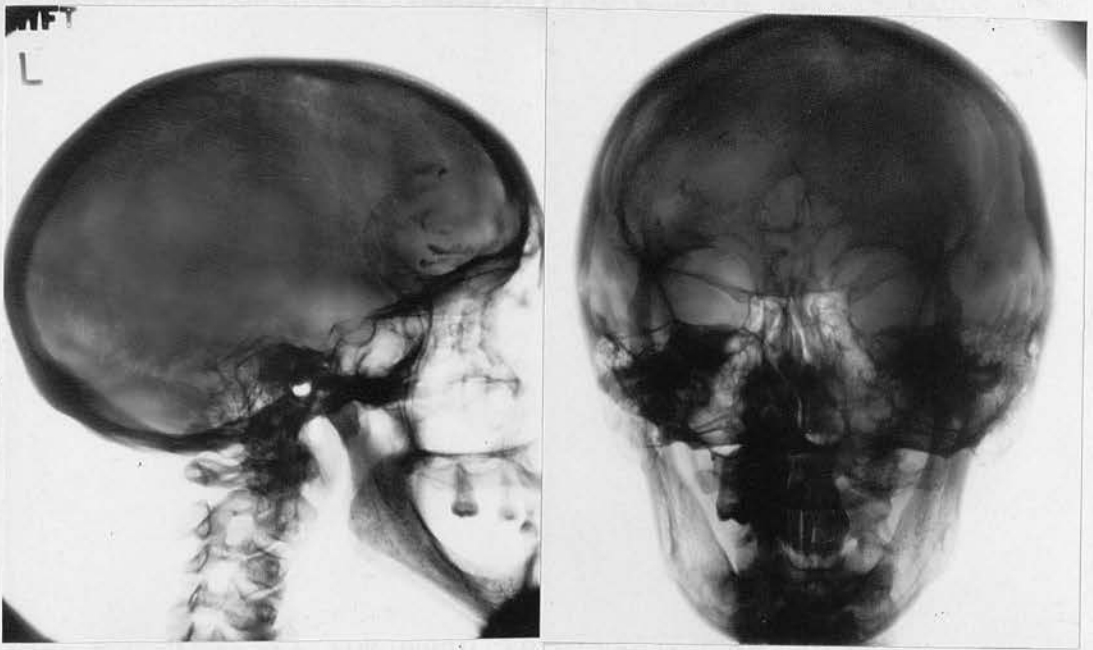


Fig. 13. Ellipse of calcification in left frontal lobe suspected of being in the wall of a chronic abscess. At operation the lesion was found to be a cystic and solid oligodendroglioma.

with compound fractures, penetrating wounds, etc., in which detailed X-ray examination is indispensable. The position of indriven fragments of bone or metallic foreign bodies is necessary information because such fragments may be the site of abscess formation.

As with other branches of radiology, the development of contrast media has proved to be an enormous advance. This applies particularly to ventriculography which at best will disclose the presence of a space-occupying lesion and indicate its precise location, without, however, giving much information as to its pathology. Our practice is to inject the air directly into the ventricles through trephine openings in the parietal region on both sides, rather than to use the indirect method (encephalography) of injecting it into the lumbar or cisternal subarachnoid space. The latter method carries definite risk to life in cases of increased intracranial pressure, and is less reliable for adequate filling of the ventricles than the direct method. Indeed, the safety of ventriculography is often questioned, but the risks appear to be no greater than in clinically comparable cases of pressure due to neoplasms, and there have been no cases in this series in which it has been felt that ventriculography has prejudiced the chances of successful treatment. There are many cases in which it

is not necessary, and there are others in extremis in which time is of vital importance and death might occur while the ventriculogram is being done. Its use calls for ordinary clinical judgement, but in cases where it is necessary the information it gives is proportionately greater than the risks involved. It should be said, however, that our practice is to follow the ventriculogram immediately by operative treatment, and the risks are considerably increased the longer such treatment is deferred. Ventriculography in cases of brain abscess, as of neoplasm, is only justifiable in circumstances which allow immediate operation, and it should be regarded as the last pre-operative diagnostic measure. It so closely approaches the stage of treatment that further consideration will be deferred to that section (p. 114).

In some cases the abscess will be encountered in the process of filling the ventricles with air - i.e., the needle may strike the abscess in its search for the ventricle. In these cases it is not necessary to proceed with the air injection, as the size and site of the abscess can be shown by aspirating the pus and instilling 2 c.c. of thorotrast into the cavity, as suggested by Kahn (18). Thorotrast is opaque to X-rays and the skiagram will show the size and extent of the lesion. It becomes fixed in the wall of the abscess,

and subsequent skiagrams will show variations in size and tension which is valuable information as to the behaviour of the abscess.

Another contrast technique which may be of value is cerebral angiography - i.e., the delineation by X-rays of a contrast medium (thorotrast) circulating in the cerebral blood vessels. In brief, the method entails the exposure of the common carotid artery between the two heads of the sternomastoid muscle, and the rapid injection of 7-10 c.c. of thorotrast into it. Serial X-rays are taken during the injection and immediately afterwards, the first exposure showing the arterial system (arteriogram) and subsequent ones the venous drainage (phlebogram). From displacements of the vessels the presence of a space-occupying lesion can be inferred (Fig. 14). In solid neoplasms the vascular pattern may be displayed and something of the pathology surmised, as some types of tumour have a characteristic angiographic appearance. In cystic tumours and abscesses there is no such vascular pattern, and this too may be of diagnostic value.

This method is employed extensively in other clinics, and there is much to be said for it. It is a simple procedure, safe, and often gives precise information. Moreover, it does not alter the pressure relationships

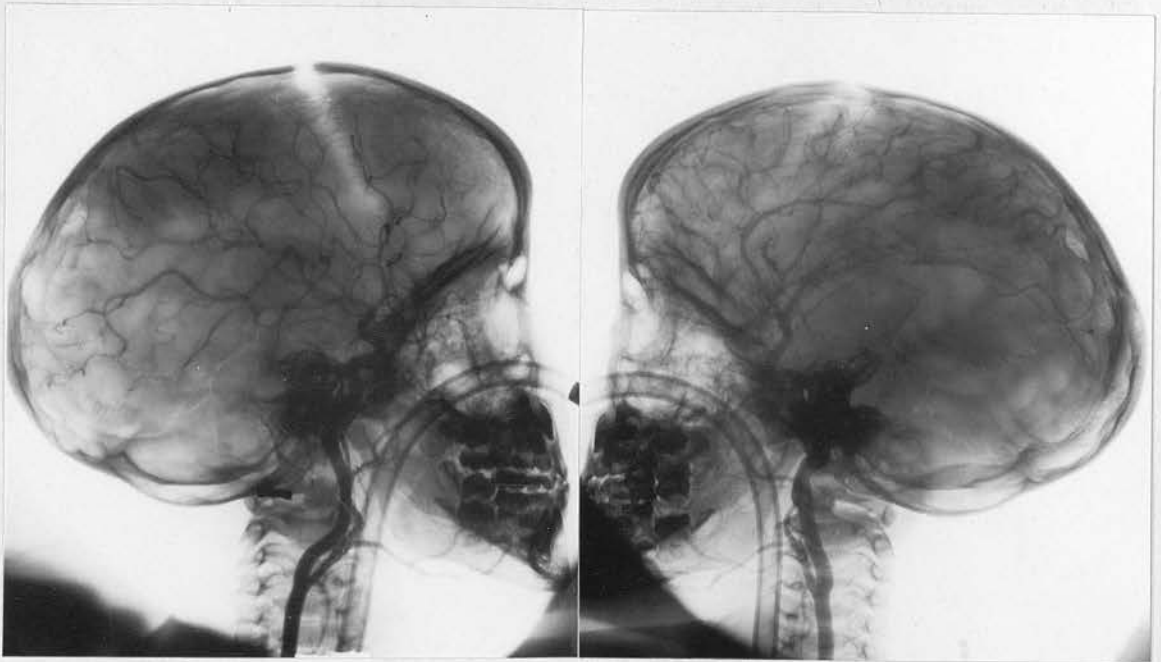


Fig. 14. Arteriogram in a case of left temporal abscess. The plate at the left shows the normal arterial pattern outlined by injection of thorotrast into the right common carotid artery. The plate on the right was taken after a similar injection into the left carotid artery. Note on this side the upward displacement of the Sylvian group of vessels, and the relative avascularity of the temporal lobe. The faint shadow of the abscess already filled with thorotrast can be seen in the anterior part of the temporal lobe, but the arteriogram suggested that there was another loculus posterior to it, and this was indeed the condition found at operation.

within the cranium, and whereas an abscess usually needs to be dealt with immediately after a ventriculogram there is not the same urgency after angiography. We have not made much use of it because ventriculography usually gives more information about the extent of intracranial lesions than does angiography, and we rely on it as the diagnostic procedure of preference in the greater part of our work which is concerned with neoplasms. Thus, an arteriogram might indicate a tumour in the right temporal lobe, but a ventriculogram would show that it also extends into the basal ganglia and is thus out of the scope of radical surgery. Angiograms in cases of slight hydrocephalus (due to cerebellar abscess or tumour, third ventricle tumour, etc.) are very difficult to interpret, whereas ventriculography usually presents no such difficulties. Perhaps the greatest use of angiography will be found in adopting it as a procedure complementary to ventriculography: the angiogram may give precise information about the presence and site of a lesion and in some cases about its pathology. When the information is incomplete or equivocal, it should be followed by ventriculography.

The Neurological Picture. The clinical examination of the nervous system in cases of brain abscess reveals signs due to increased intracranial pressure and to a focal lesion of the brain. One or the other group of

signs may predominate or exist almost to the exclusion of the other: thus, in Cases 1 and 4 (bronchogenic abscesses) there were crippling hemiplegias at a time when the patients had very little headache and no papilloedema; on the other hand, in some chronic abscesses (Cases 15 and 18) there may be intense papilloedema with practically no other neurological abnormalities.

The general symptoms and signs of increased intracranial pressure have been recounted (pp. 52-61): headache, stupor, incontinence of urine and faeces, slowness in chewing and swallowing, depression of respiration are all common features. Objective evidence of increased pressure is afforded by the presence of papilloedema, elevation of the spinal fluid pressure, and, in chronic cases, by skiagrams which show convolutional thinning of the vault of the skull and erosion of the dorsum sellae. In children there may be separation of the sutures which can be seen in the skiagrams and can be inferred clinically by finding that the skull is enlarged and has a high-pitched **or** cracked-pot note on percussion. In some cases in childhood (as in Case 18, a frontal abscess in a child of six years) there is a loud bruit audible all over the skull: this is a common feature of increased intracranial pressure in childhood, and it has no special diagnostic significance. Another common sign of increased pressure is paralysis of one or other

abducens nerve to produce a squint and diplopia. This too occurs in increased pressure due to various causes and is of no localising significance.

These general signs of increased pressure are, of course, not peculiar to brain abscesses: they differ in no way from the same signs due to neoplasms, haematoma, obstructive hydrocephalus, etc. They are generally gross and require no special skill on the part of the examiner to elicit them. They can be demonstrated with little or no co-operation of the patient and thus form the most primitive part of the examination. On the other hand, a detailed examination of the nervous system does require the conscious co-operation of the patient, and anything short of this may greatly tax the patience of the examiner. The information gained is well worth the effort, however, and every case should be examined as fully as circumstances permit. An early examination is valuable not only because the patient is still able to co-operate, but it establishes a base-line for the assessment of subsequent developments. Thus an abscess may be suspected and the progression of neurological abnormalities in repeated examinations will at least show that there is a progressive intracranial lesion. Too often cases are left without an adequate examination until deep stupor or coma supervenes, and

then the diagnosis and localisation become much less accurate. In such cases time is of vital importance, and the loss of time entailed in special diagnostic procedures (which might not have been necessary had the patient been examined earlier) may influence the outcome adversely.

As an abscess of the brain may produce almost any neurological abnormality, I propose to deal with the problems of localisation in regional categories - i.e., describing the signs of abscess in the frontal and temporal lobes, in the cerebellum, and elsewhere in the brain, as this is usually the way in which the problem is presented to the clinician.

Abscess of the Frontal Lobe (see Cases 1, 4, 14-18). The onset of a frontal abscess due to sinus infection has been discussed above (p. 46 et seq.). An epileptic attack occurred in the majority of the cases in this series, and when a description of the fit was available there was evidence to lateralise the lesion. This is valuable information because there may be little else to tell in which frontal lobe the abscess is situated: the headache may be generalised, and the superficial signs of inflammation may be in the centre of the forehead; and as an acute infection of the frontal sinus usually involves

the whole sinus the intracranial extension may take place in either frontal lobe. If the epileptic attack is characterised by turning of the head and eyes to one side, with involuntary movement of the limbs on that side, it is reasonably certain that the lesion is in the opposite frontal lobe. In one case (Case 14) there were no such "adversive attacks", but there were focal motor attacks involving the opposite side of the face without loss of consciousness. There may be a transient weakness of one side of the face or the limbs involved in such an attack, and although this may clear up completely within a few hours recovery may be not quite complete and the weakness becomes progressively more marked as time goes on. But in only one case in this series was the weakness at all profound (Case 14); in all the others it was slight, by no means disabling, and usually the patients were not even aware of it. In these cases, however, there were significant alterations in the reflexes, the tendon reflexes being slightly increased on the affected side, the abdominal reflexes diminished or absent, and the plantar response extensor. In none of the cases were any sensory abnormalities detected.

In two cases, the abscess was in the speech-dominant hemisphere. In one of them there was a slight expressive

aphasia, but in the other no disturbance of the language function could be demonstrated. "Mental symptoms" likewise were not marked: in Case 14, an acute abscess, there was some delirium and confusion, but these symptoms may have been due as much to the general effects of the infection as to the location of the lesion. The most profound mental disturbance in the whole series occurred in the case described on p. 49, and the lesion in this case was in the lower part of the Rolandic region, if anything more in the parietal than in the frontal lobe. We have noticed that some patients with frontal abscess are rather quiet, somewhat sullen and suspicious, but not enough to influence their behaviour to any great extent, and the same features have been observed with lesions in other parts of the brain.

Frontal abscesses thus may be remarkably silent as regards neurological abnormalities. This very silence is a danger and it should be said again that any case of frontal sinus infection which has shown any of the signs described above should be kept under observation until it is clear that the infection has resolved and that there is no chronic intracranial suppuration. If, as the sinus infection resolves or responds to treatment, the patient improves generally, becomes free from symptoms and signs, has a normal spinal fluid pressure

and normal fluid on analysis, it can be assumed that there is no continuing trouble in the brain. In some cases the spinal fluid findings may be equivocal: the pressure on the high side of normal, with perhaps a protein content on the high side of normal too. The appearance of the optic fundi may be suspicious, or there may be frank papilloedema without any other symptoms or signs. In these cases the only method of being certain as to whether or not an abscess is present is ventriculography: if there is no abscess the ventriculogram will be normal; if there is an abscess there will be a slight displacement of the ventricular system to the opposite side and deformity of the frontal horn on the side of the lesion (Fig. 15).

Frontal abscess due to causes other than sinus infection are more eloquent the farther back in the frontal lobe they are situated. Part of the silence of the frontal sinus abscess is due to the fact that it develops at the frontal pole, a notably silent area of the brain. Similarly, in Case 18, a polar abscess which eroded the roof of the orbit and caused proptosis, there was a remarkable absence of neurological signs. On the other hand, in Cases 1 and 4, blood-borne abscesses in the Rolandic region, there were repeated Jacksonian attacks and profound hemiplegia. In Case 16, an abscess in the posterior part of the right frontal lobe due to a compound

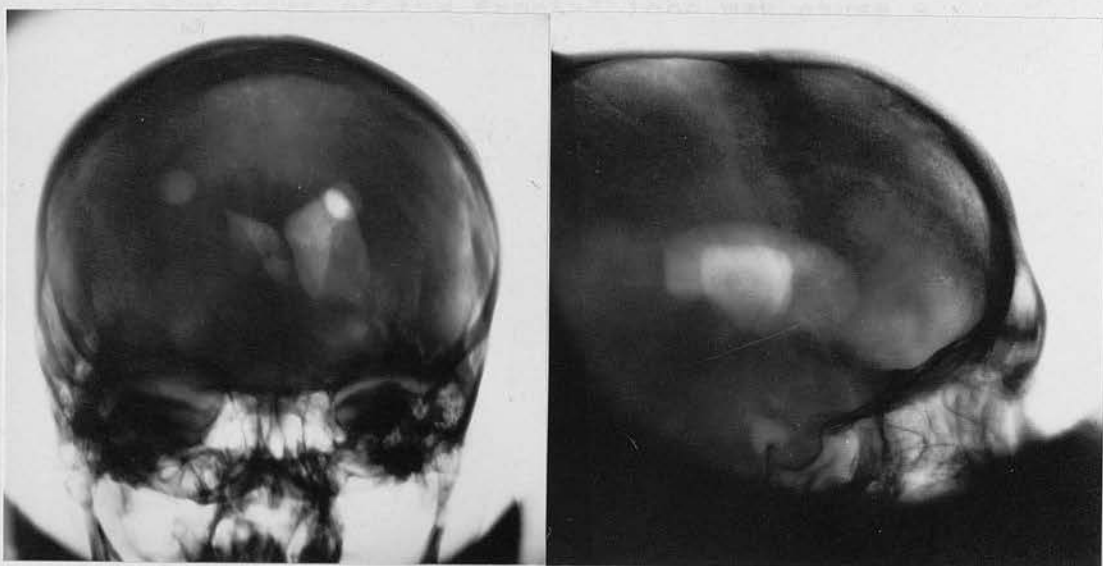


Fig. 15. Ventriculogram of right frontal abscess due to acute frontal sinusitis. Note shortening of right frontal horn and displacement of ventricular system to the left side.

fracture, there was a complete paralysis of the left arm and leg. In the speech-dominant hemisphere an abscess in the posterior part of the frontal lobe may cause a gross disturbance of the language function, and if the lesion is in the region of the operculum insulae there may also be a characteristic slurring dysarthria.

Abscess of the Temporal Lobe (Cases 8-11, 19, 20). The most constant and characteristic feature of an expanding lesion in the temporal lobe is a defect in the opposite homonymous field of vision. That part of the geniculocalcarine pathway which subserves the upper quadrant of the opposite half-field runs forward in the temporal lobe and bends back around the temporal horn before passing back to the occipital cortex (Fig. 16). An interruption of these fibres produces a defect in the upper quadrant of the opposite field, and as the lesion expands to compress or destroy the rest of the optic radiation a complete hemianopia may result. In all cases of temporal abscess in this series in which it was possible to assess the visual fields, some degree of this defect was demonstrated. It may be so slight as to be apparent only on careful perimetry, and this may be the only demonstrable neurological abnormality. Thus, one patient with a persisting increase of intracranial pressure after a mastoid operation was regarded as suffering from otitic hydrocephalus

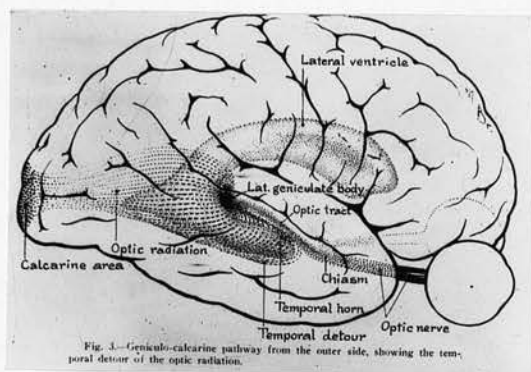


Fig. 16. Schematic representation of the geniculo-calcarine pathway (from Cushing and Meyer). The lower fibres of the radiation, subserving the upper quadrant of the ~~opposite~~ half field are seen sweeping down around the temporal horn of the ventricle before coursing back to the calcarine cortex. This "temporal detour", so-called by Meyer, is thus likely to be involved in expanding lesions situated in the middle of the temporal lobe, and as this is a common site for otogenic abscesses, the frequency of upper quadrant hemianopia is to be expected.

because of his good general health and the absence of any neurological abnormalities except marked papilloedema. The visual fields were full to confrontation tests, but when they were tested carefully on the perimeter and Bjerrum's screen a definite upper quadrant defect was demonstrated. (Fig. 17) and the diagnosis of right temporal abscess was confirmed at operation. Accurate perimetry demands the co-operation of the patient, and this is an additional reason for having him under observation before stupor and inattention become marked. The visual fields should be tested carefully in every case of mastoiditis which is not running a normal course.

If the abscess is in the temporal lobe of the speech-dominant hemisphere some degree of aphasia is usually present. This may be manifest in a difficulty of naming common objects, inability to write correctly, or to read or to understand the spoken word. Gross aphasia is easy to recognise, but slight defects may go unnoticed unless these simple tests are carried out. If the patient is too deeply stuporose to speak coherently or to co-operate in formal tests, enquiry from his relatives may disclose some disturbance of speech or writing which, taken with other evidence, may be of diagnostic and localising value. In young children a common story is that they have "stopped talking", and in older children an unwillingness to talk is a frequent manifestation of aphasia: the

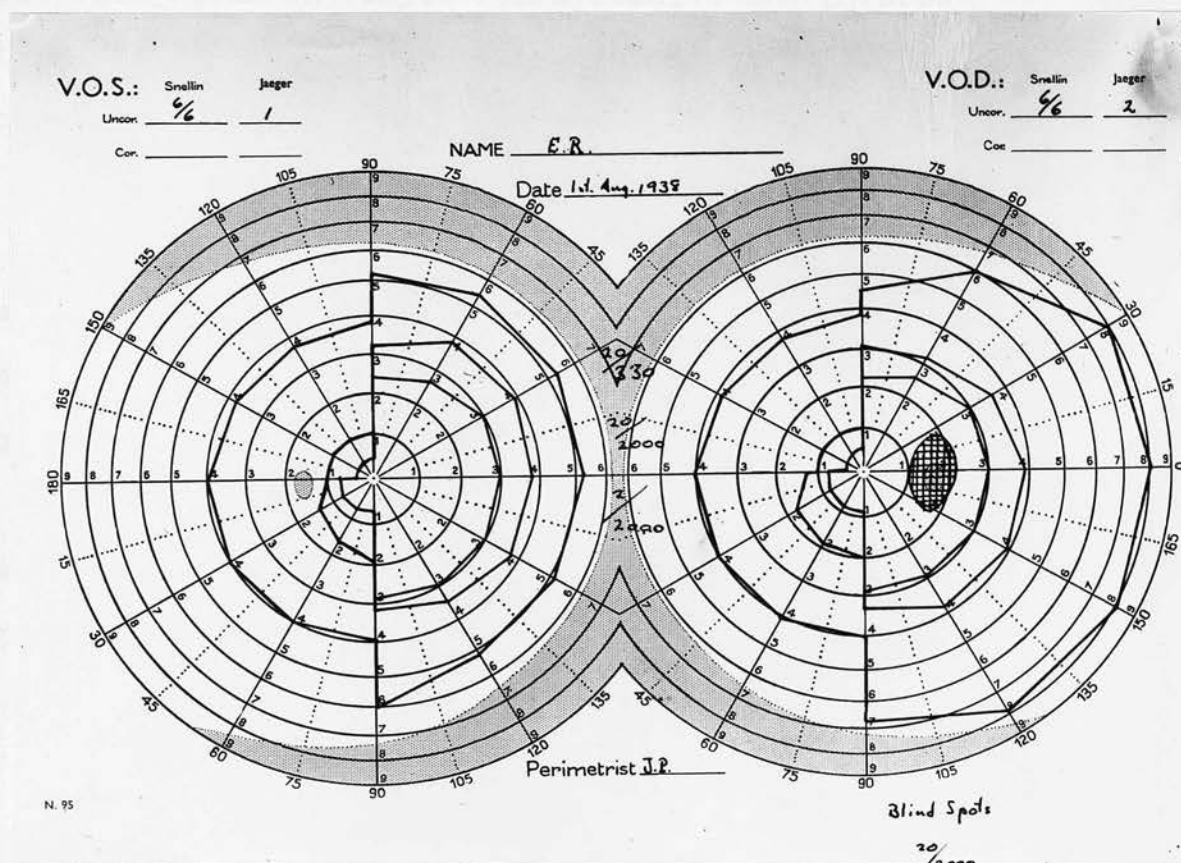
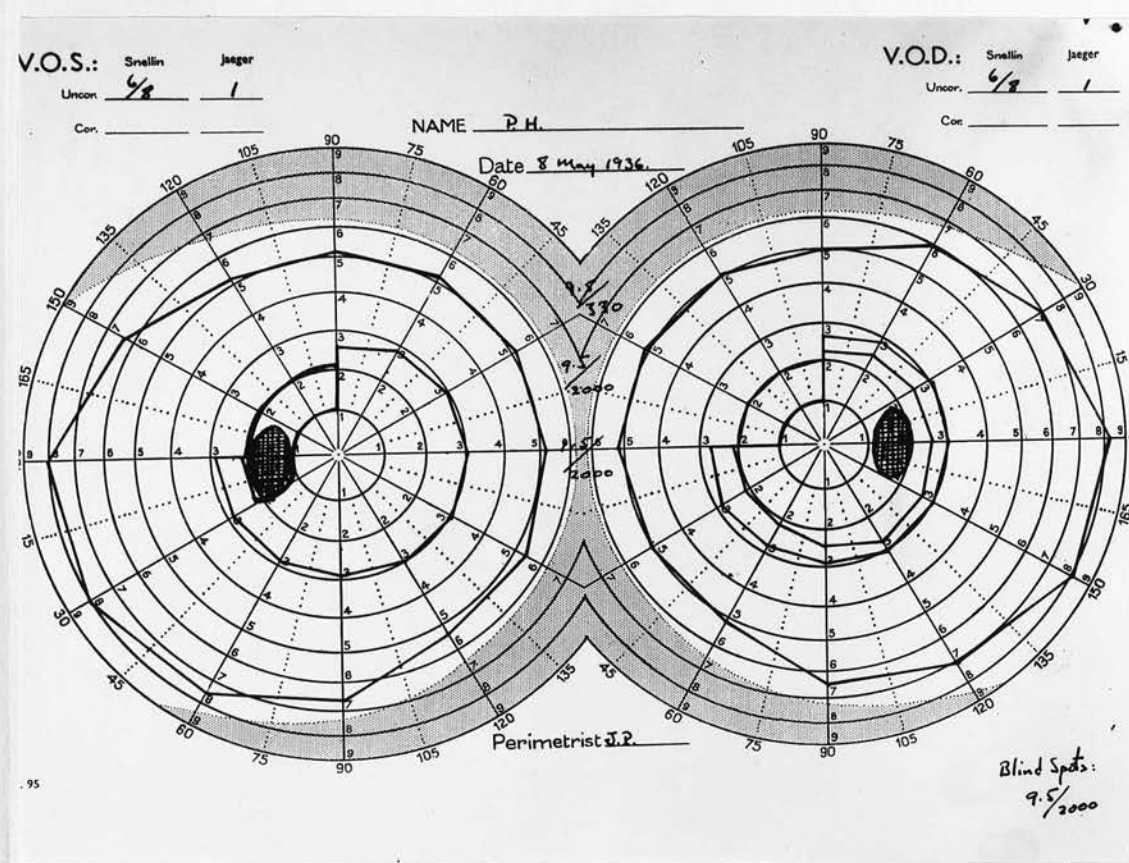


Fig. 17. Visual field charts in a case of right temporal abscess. Although a hemianopic defect, it is most marked in the upper quadrant. The defect was not detected on confrontation tests.

This is the case mentioned in the text in which the diagnosis of otitic hydrocephalus was made until the field defect was demonstrated by perimetry. Additional evidence for the diagnosis was afforded by the fact that the cerebrospinal fluid contained an increase in protein and cell content on repeated examinations.



Visual field chart in another case of right temporal abscess due to pre-existing mastoid disease. The defect was not apparent on confrontation tests, and perimetry demonstrated a sharp upper quadrant defect in the left homonymous field. This was the only definite objective sign, and it led to the diagnosis of a right temporal lesion which, from the history of the illness having started with an acute otitis media, was presumed to be an abscess. This diagnosis was confirmed at operation.

child will speak in simple monosyllables and phrases rather than in more complicated sentences, almost as though they were ashamed or afraid of being "caught out" in an error of speech.

Although aphasia is a common sign of temporal lobe lesions, it may occur with lesions in any part of the speech-dominant hemisphere, and especially in the vicinity of the Sylvian fissure. It has been mentioned as occurring in lesions of the frontal lobe, and it is also common in parietal and occipital lesions. Psychologists have attempted to analyse the language function in terms which would give localising value to particular types of aphasia, but the more carefully this function is analysed the less pure any type of aphasia becomes, and about all that can be said is that the kinds of aphasia which are predominantly receptive (sensory) are apt to be due to lesions in the vicinity of the posterior part of the Sylvian fissure (e.g., supramarginal gyrus), while the expressive types (motor aphasia) are due to lesions farther forward, e.g., in the posterior part of the frontal lobe.

In a small proportion of cases, as in one of this series, an abscess of the speech-dominant temporal lobe does not produce aphasia. These exceptions also occur in cases of neoplasm and vascular accidents, and are

probably to be explained on the basis of crossed hemisphere-dominance or of ambivalence. Aphasia thus is a useful lateralising sign when it is present, but its absence does not exclude a lesion in the dominant hemisphere.

Apart from visual field defects (which may be very slight), and aphasia (which only occurs if the lesion is in the dominant hemisphere) the temporal lobe is one of the notoriously silent areas of the brain. Pressure signs (intense headache, stupor, sixth nerve palsy, etc.) may occur early and be the striking feature before there is any gross disturbance of motor power or of sensation. One explanation for this is that expansion of the temporal lobe forces the uncus down through the incisura tentorii and obstructs the circulation of the cerebrospinal fluid. This medial pressure may also involve the oculomotor nerve and produce unilateral ptosis, dilatation of the pupil, loss of inward, upward and downward movement of the eyeball, and a divergent squint at rest (Case 19). When muscular weakness occurs it is usually most marked in the opposite side of the face, next in the upper limb, and least in the lower limb. This order of the development of hemiplegia is characteristic of temporal lobe lesions, and weakness of the face and upper limb may be very profound before there is any demonstrable weakness

or alteration of reflexes in the lower limb. It is explained by the proximity of the temporal lobe to the cortical areas and cortico-spinal fibre tracts subserving these parts. Disturbances of sensation usually occur when there are motor abnormalities because of pressure on the lower part of the post-central gyrus and the lower part of the corona radiata. These signs likewise appear in the face, upper limb, and lower limb, in that order, and as with most cortical and sub-cortical lesions the disturbance is much less obvious than a motor deficit of the same degree. Thus, in the case described on p. 49 removal of the abscess entailed the excision of the lower part of the right post-central gyrus and the subjacent white matter superficial to the ventricle. After the operation this patient could appreciate minimal stimuli with cotton wool on the left side of the face and left upper limb, although tactile localisation was slightly defective: he would fail to identify the spot touched by 2-3 cm. Pin-prick had a "tingly" quality, and the threshold for identifying the prick as painful was raised as compared with the right limb. This and other forms of painful stimuli had a diffuse quality, seeming to affect a larger area of skin than the actual spot stimulated. Although he could feel light touch and identify pin-pricks as sharp, there was a complete loss of postural

sensibility to the point of his "losing" his arm in bed, and of stereognostic sense, and of the ability to differentiate between one and two points of a compass. Such profound degrees of sensory loss are not common in temporal lobe lesions, but lesser degrees will be found in many cases on careful examination.

As the sensory and motor abnormalities are usually the result of pressure on, rather than destruction of, the relevant neurones, they usually disappear entirely when the abscess has been successfully treated. Aphasia likewise usually clears up: slight defects may persist, but in no case in this series has the residual defect been at all disabling. Defects in the visual fields, on the other hand, are often permanent either because of destruction of the fibre-tracts by the abscess or because of operative trauma. An upper quadrant defect usually occasions very little inconvenience, but a complete hemianopia may be troublesome for the patient until he becomes adjusted to it. A left homonymous hemianopia is usually more of an inconvenience than one to the right side because of the difficulty in finding the beginning of the next line in reading.

Abscess of the Parietal Lobe (Cases 5 and 7). Abscess of the parietal lobe usually results from a blood-borne infection or from trauma. As the parietal lobe is

supplied by the middle cerebral artery it is a common site for the lodgement of infected emboli. Focal epileptic attacks may signify the onset of the infection and these are characterised by paraesthesiae in one side of the face, or in one arm or leg. These sensory phenomena may constitute the aura of a motor attack of the Jacksonian type, as frequently happens with lesions in the Rolandic region. There are often residual defects in sensation which **have** the characters of the "cortical type" of sensory loss as described on p. 83 - i.e., with little disturbance of tactile sensibility, but gross loss of postural and discriminative modalities. There may also be visual field defects: characteristically a lesion in the posterior part of the parietal lobe produces a defect in the lower quadrant of the opposite homonymous field, but lesions in the anterior part (e.g., post-Rolandic) may not affect the visual pathway at all. Although the parietal lobe is largely concerned with sensation, as the lesion expands the motor pathway is involved, and varying degrees of hemiplegia are common. Indeed, the hemiplegia may be more obvious than the sensory loss unless a careful sensory examination is made. Aphasia may occur if the lesion is in the speech-dominant hemisphere and especially if it is in the lower and posterior part of the parietal lobe. If the aphasia has any

characteristic feature it will probably be that it is largely of the receptive type - e.g., difficulty in understanding the written or spoken word.

A peculiar effect of parietal lesions which has been encountered in two cases in this series is a disorder of spatial orientation. This manifested itself in a striking difficulty of the patient's finding his way about in well-known surroundings. In one case (Case 5) it was an early symptom: the patient got lost at her work in a telephone exchange and had to be shown the way to her home and even to her own bedroom. In the other case this disturbance was noticed during convalescence after the destruction of a large part of the parietal lobe by a metastatic abscess. This patient had the greatest difficulty in finding his way about the ward and frequently got lost in the hospital grounds despite the fact that his visual acuity was normal and his intellect unimpaired. He had a complete homonymous hemianopia, but as other patients with hemianopia due to other causes do not show this defect it seems that the disorder of disorientation is probably a factor of the parietal lobe. Just as the patient with loss of postural sensibility in a limb may "lose" the limb in bed, so he may lose himself in relation to his surroundings. This phenomenon has been described by other authors, and Brain (19) has recently reviewed the subject.

Abscess of the Occipital Lobe. Abscess of the occipital lobe is usually due to trauma or to an extension from a primary infection of overlying parts of the scalp or skull. The occipital lobe is largely supplied by the vertebral circulation and it is less commonly the site of embolic abscesses than those parts of the brain supplied by the internal carotid artery. Mention has already been made (p. 9) of bilateral occipital abscesses due to bronchiectasis; and in cases of generalised pyaemia abscesses may develop in any part of the brain. The frequency of abscesses in the middle cerebral distribution and their rarity in the posterior cerebral distribution suggests that in the former it is a matter of the lodgement of infected emboli rather than the mere presence of organisms in the blood-stream which presumably would be distributed evenly between the carotid and vertebral circulations.

A unilateral occipital abscess may be remarkably silent: hemianopia in the opposite homonymous field is the most characteristic sign, but if it develops slowly the patient may not be aware of it. Attention may be drawn to the visual pathway by the occurrence of visual hallucinations (teichopsiae, fortification spectra) which can be looked upon as focal epileptic phenomena equivalent to the focal motor or sensory attacks which

occur with lesions of the Rolandic region. These hallucinations take the form of flashes of coloured lights, scintillating sparks or coloured geometrical patterns, the so-called "unorganised" visual hallucinations. They may constitute the aura of a generalised epileptic attack with loss of consciousness, in which case the patient may forget to mention the aura, or he may not even remember its occurrence on direct enquiry. Even if there are no other components to the attack, the experience may be so alarming that he cannot analyse it, but if he can do so he may be able to say that the hallucination occurred in one or other homonymous field. In any case, the hallucinations are generally followed after a short time by the development of a homonymous hemianopia. As the abscess expands it may produce pressure on the parietal lobe and the internal capsule to cause contra-lateral sensory defects and hemiplegia.

Bilateral occipital abscesses are rare, but they may occur by simultaneous lodgement of infected emboli in the distribution of both posterior cerebral arteries. Such lesions may cause the sudden onset or rapid development of bilateral blindness without any change in the optic fundi. As the lesion is above the geniculate bodies (i.e., nearer the cortex) the reaction of the pupils to light may be preserved even though the patient has no conscious perception of light.

Cerebellar Abscesses. Seven of the eight cases of cerebellar abscess in this **series** were due to mastoid infection, and in all except one of these (Case 13) the relation to the mastoiditis was so clear that the diagnosis presented no unusual difficulties. The occurrence of severe headache during a chronic mastoiditis or following a mastoid operation was invariable in these cases, although in one the mastoid infection appeared clinically to be a mild one which might have resolved without operation. The character of the headache and its effect on the posture of the head have been discussed above (p. 53). Vomiting is a frequent occurrence and as an early symptom is commoner in cerebellar abscesses than in lesions above the tentorium. Dysarthria is a common sign too: the speech becomes thick and slurred, probably due to inco-ordination of the muscles of articulation, as can be demonstrated by getting the patient to perform rapid alternating movements with the **tongue**. In one case of bilateral mastoid disease the speech disturbance was at first thought to be a manifestation of aphasia, in which event the evidence would have favoured localisation in the left temporal lobe. Closer study showed that there was no disturbance of the language function and that the defect was simply a matter of dysarthria: this suggested that the abscess

was in the posterior fossa, and at operation it was found in the right cerebellar lobe. Dysarthria occurs before dysphagia, although in the later stages there may be difficulty in swallowing too.

The neurological abnormalities produced by an abscess in one cerebellar lobe are generally easy to elicit and often demand only to be looked for. Characteristically there is nystagmus which is slow and coarse when the patient is asked to look toward the side of the lesion and more rapid and finer when the eyes are deviated to the opposite side or upward. In some cases the nystagmus may be accompanied by a "skew" asymmetry of the visual axes on lateral deviation of the eyeballs. A common type of this asymmetry can be described as follows: instead of the eyeballs maintaining the same horizontal plane, the abducting eye is directed so that it looks below the horizontal, while the adducting eye looks above the horizontal, and the patient complains of a corresponding diplopia. Another abnormality of the external ocular movements which is often seen but is not peculiar to cerebellar abscesses is limitation of conjugate upward movement of the eyeballs. This may be associated with slight bilateral ptosis and sluggish reaction of the pupils to light. It occurs in advanced stages of increased intracranial pressure, and is probably due to

pressure effects on the mid-brain by structures herniated through the incisura tentorii (p. 17).

From their common situation **in the** anterior part of the lateral cerebellar lobe, it might be expected that cerebellar abscesses would occasionally simulate tumours of the cerebello-pontine angle in their effects on the cranial nerves in the vicinity. It has been mentioned that paralysis of the sixth nerve is common in cases of increased intracranial pressure due to various types of lesions in various situations, and it is of no localising value. Sensory defects in the trigeminal distribution do occur, but are rare in abscess, whereas they are very common in cerebello-pontine angle neoplasms. Slight degrees of impaired sensation were present in two cases in this series, but they were very slight and amounted only to a subjective difference in the quality of the sensation in the two sides of the face and slight depression of the corneal reflexes.

Facial weakness is more common than trigeminal impairment, and was present in four cases. It too occurs less constantly in abscess than in neoplasm, but the characters of the weakness are essentially the same. The weakness is on the same side as the lesion and is of the nuclear or peripheral type: all segments of the face are affected to an equal extent, and the weakness is as

pronounced in voluntary as in emotional movements. It may be very slight, but a common observation is that there is delayed and incomplete lid-closure during blinking on the affected side of the face. This asynchronous blinking may be apparent before it is possible to demonstrate any definite weakness in formal tests, such as closing the eyes, showing the teeth, etc. In one case we have seen fibrillation in the affected facial musculature, and the phenomenon of mass innervation: i.e., the patient is unable to innervate one part of the face without the whole contracting. When he is asked to screw up his eyelids the angle of the mouth retracts; and when he is asked to show his teeth the lids and brow contract too. These are features of a slowly-developing facial paralysis or of recovery, as after a Bell's palsy, and are much commoner in cases of neoplasm than of abscess. They are of value in indicating pressure on the facial nerve in some part of its course, or recovery of a pre-existing paralysis.

It is difficult to assess the deafness in a case of cerebellar abscess because of the pre-existing mastoid disease and operative procedures. Usually it is of a mixed type and rarely is it quite complete. In some cases (as Case 12) there may be no deafness at all, and in others it may be very slight. Tests of vestibular

function usually are referable more to the mastoid disease than to the abscess: in the cases in which the patient's condition has permitted caloric tests to be done the response was usually present, but delayed, and quantitatively less **than** on the normal side. We have not found that these tests contribute very much to the diagnosis in cases of abscess, although they are invaluable in the case of neoplasms, an absent caloric response being the most constant sign of an acoustic neurinoma, for instance.

Effects on the lower cranial nerves may contribute to the dysarthria and dysphagia described above by causing weakness of the **palate**, tongue, pharynx, and larynx. Gross paralyses are rare, and it is probable that the disturbances of speech and swallowing are more largely due to inco-ordination than muscular paralyses.

The most characteristic disturbance of motor function is inco-ordination. In the acute cases (as in Case 12) there may be wild ataxy of the arm and leg on the side of the lesion, while in the chronic cases (Case 13) the defect may be very slight and only demonstrable on formal tests, e.g., the finger-nose-finger and the heel-shin-finger test. This difference between slowly-growing and rapidly-growing lesions is also seen in cases of neoplasm: a massive cerebellar astrocytoma may produce

very little evidence of cerebellar dysfunction, whereas a rapidly-growing tumour or one associated with cyst formation usually produces gross signs. The cerebellum seems to have a remarkable capacity for adapting itself to expanding lesions if they develop slowly, but it is always surprising to see a tumour which occupies nearly the whole of the posterior fossa and has produced almost no cerebellar signs.

Associated with the ataxy there is usually hypotonia and dysdiadokokinesis, and slight muscular weakness. The tendon reflexes may be diminished as compared with the normal side, but the abdominal and plantar reflexes are normal unless there are indirect pressure effects on the pyramidal system by increased intracranial pressure, in which case there may be increased tendon reflexes and extensor plantar responses. Local compression of the ipsilateral pyramidal tract in the brain stem will cause spasticity and weakness of the opposite limbs, with corresponding alterations in the reflexes. In such cases there are cerebellar signs on the side of the lesion and pyramidal signs on the opposite side.

If the patient is ambulant, there may be unsteadiness of gait, and in some cases (Case 13) this may be the most impressive manifestation of inco-ordination. The patient walks on a wide base, may give an occasional

lurch to the side of the lesion, especially in turning around quickly, and commonly he does not swing the arm on the affected side as freely and as naturally as on the normal side. This unsteadiness in walking is a sign of cerebellar dysfunction, and is usually not associated with any defect of sensation (as in tabes and subacute combined degeneration) or with giddiness. Giddiness occurs frequently in cerebellar abscesses - and it may be either true vertigo or a less well defined feeling of light-headedness. But this is a common symptom of mastoid infection and of increased intracranial pressure, and we have not found it of particular value in the symptomatology of cerebellar abscess. Although it may occur in patients with gait disturbances, the gait may be unsteady even when the patient is experiencing no giddiness at the time. On the other hand, an attack of vertigo or of light-headedness will accentuate a gait disturbance to such an extent that the patient has to stop or fall down until the attack passes off.

Before leaving the question of ataxy, it should be said that it is possible to mistake ataxy for paralysis in children or unco-operative adults. We have often noticed that a child is very loath to use an ataxic limb and may allow it to rest immobile in bed, giving the appearance of paralysis. Moreover, they may be unwilling

to co-operate in tests for co-ordination, although quite co-operative in other respects. Just as they seem to be ashamed or afraid of being "caught out" in tests for aphasia, they exhibit the same reaction in tests of cerebellar function. One little boy of 5 years would be quite cheerful in performing the finger-nose-finger test with his normal limb, but wept bitterly and shamefacedly when he was made to perform the test with his ataxic limb. The semblance of paralysis may be continued until the patient is seen trying to adjust the bedclothes or reaching for something on his bedside table when he does not know that he is observed. The distinction is of obvious importance because it may differentiate a lesion below the tentorium from one above it.

VI. Differential Diagnosis.

In this clinic we have an index system relating to differential diagnosis. It deals with the many hundreds of cases of intracranial disease which find their way to a neurosurgical clinic, and although such an index can never be precise it is a useful guide in the analysis of specific problems of differential diagnosis. Reference to this index shows that the following conditions were apt to be mistaken for brain abscess, in this order of frequency:

1. Other expanding lesions (e.g., neoplasm, subdural haematoma).
2. Intracranial venous obstruction ("otitic hydrocephalus", cerebral thrombophlebitis, traumatic sinus thrombosis).
3. Other intracranial infections (subdural abscess, extradural abscess, purulent leptomeningitis, tuberculous meningitis, petrositis).
4. Arterial hypertension.
5. Psychoneurosis.

It is not difficult to see that the most common problem of differential diagnosis is that of distinguishing between an abscess and other expanding lesions such as neoplasms and subdural haematoma. This difficulty arose most frequently among the cases in Group C. of the metastatic abscesses (Table I, p. 6), i.e., those due to infection from sources other than the thorax. In 6 of the 7 cases in this group the lesion was thought to be a neoplasm until it was disclosed at operation. The importance of the differentiation is that of the 5 cases operated on (one is under treatment at present) all made good recoveries and are leading useful lives.

The same difficulty was encountered occasionally in other types of abscess: there was indeed one case in each of the other groups in which the pre-operative diagnosis was "abscess or tumour". These were cases in which the primary infection (of the lung, mastoid, frontal sinus, etc.) had resolved and a considerable interval had

elapsed before the brain abscess declared itself.

The diagnosis of abscess of the brain depends on the knowledge of a primary source of infection, present or pre-existent. If the patient is too drowsy to relate such an infection, and if his relatives have no knowledge of it, we may have to rely on objective evidence. The routine investigation of any case of increased intracranial pressure includes an inspection of the ears (mastoid scars, latent discharge, perforation of the tympanic membrane), clinical and radiographic **examination** of the chest (which may reveal a chronic infection or a neoplasm), X-rays of the skull (which may show an opaque foreign body, an old depressed fracture, a calcified neoplasm, evidence of increased intracranial pressure), and a general search of the skin for the scars of recent boils or carbuncles. But the most valuable evidence is afforded by examination of the cerebrospinal fluid, in which the significant abnormalities are a slight increase in the protein content and a mild pleocytosis.

These **special** investigations may give a clue to the pathology, but there are many cases in which the evidence is equivocal and the pre-operative diagnosis can only be presumptive. For example, chronic mastoiditis is common and neoplasms of the brain are common; not infrequently the two conditions occur in the same patient, just as

many patients with brain tumour have had other antecedent infections which might have produced an abscess of the brain. In the same way, many patients with increased intracranial pressure due to abscess or neoplasm can recall a head injury prior to the onset of symptoms which might have produced a chronic subdural haematoma.

It has been shown above that the symptoms and signs of increased intracranial pressure and of the focal lesion in the brain due to an abscess are indistinguishable from those due to neoplasm. If the lesion is a neoplasm the rapidity of the history usually suggests that it is a rapidly-growing one, e.g., spongioblastoma multiforme, or a less malignant type associated with cyst formation. It is common knowledge that such neoplasms are usually incurable, and indeed in many cases operative treatment is contra-indicated. On the other hand, every case of brain abscess is potentially curable, so the differentiation is a matter of vital importance. It can only be made with certainty by verifying every lesion before dismissing it as hopeless, and that is the present practice in this clinic. In the case of a rapidly-growing glioma the clinical evidence and a ventriculogram will usually give precise information as to the site of the lesion, and a biopsy is then taken through a suitably-placed burr hole. A fragment of tissue is aspirated in a hollow

needle (brain cannula) and stained with toluidin blue by the method described by Dudgeon (20) and developed especially for the nervous system by Russell (21). This procedure will give sufficient information for the pathological diagnosis in about 90 per cent. of cases. In the case of an abscess the exploring needle will strike pus or tissue of an inflammatory nature in the vicinity of the abscess, and suitable measures can then be taken to deal with it. The value of this diagnostic method has not been sufficiently appreciated, as in this clinic it has disclosed many curable lesions (abscesses and benign neoplasms) which on the clinical evidence had been regarded as hopeless.

The same procedure will lead to the detection of a chronic subdural haematoma. The haematoma may be encountered in the parietal region at the time of making the burr holes for the ventriculogram, but that is not always the case, as the lesion may be farther forward. If so, the ventriculogram is characteristic in showing a general displacement of the whole ventricular system toward the opposite side without any specific deformity or filling defect (Fig. 18). A burr hole in the frontal region will then disclose the lesion and enable it to be dealt with.

Intracranial Venous Obstruction. It is well known that obstruction to the venous return from the brain

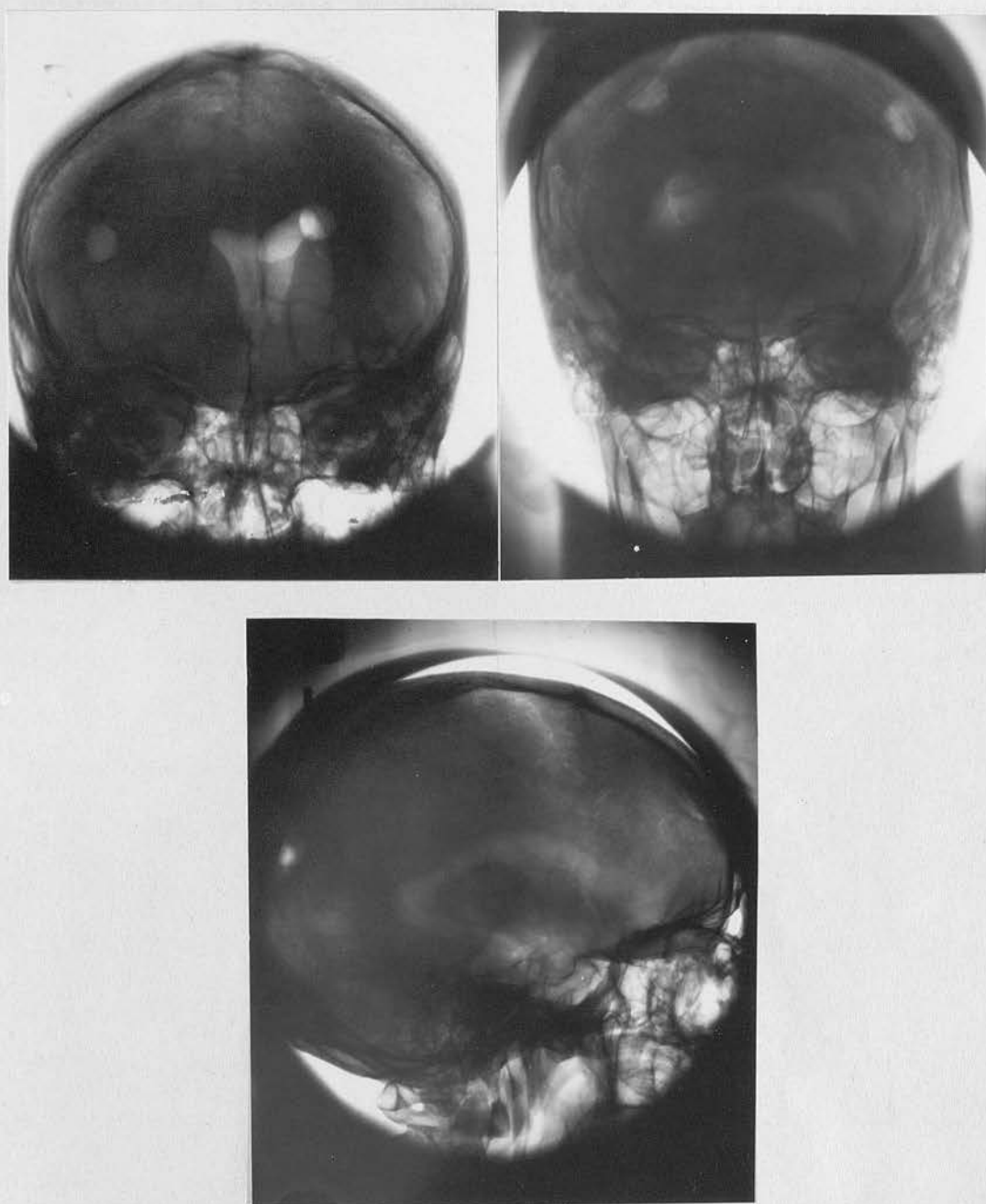


Fig. 18. Ventriculogram of right subdural haematoma, situated in the frontal region and not encountered in making the parietal burr holes for the ventriculogram. Note the absence of specific deformity and the slight general displacement of the ventricular system.

causes an increase in intracranial pressure. If the obstruction is due to thrombosis of large sinuses, there are commonly the general signs of increased pressure: headache, vomiting, papilloedema, and sixth nerve palsy. If the thrombosis extends to the cerebral veins, there may be focal neurological signs such as aphasia, hemiplegia, and defects in the visual fields. As yet, more is known about the aetiology of this condition than its pathology: as it is usually not fatal, there is little necropsy material and a good deal of the clinical picture is based on inference. Only recently has the problem been approached experimentally, by Russell and Beck (22).

The commonest type of this condition is that described by Symonds (23) in 1931 as "otitic hydrocephalus" because of its frequent association with mastoiditis. In a brief review of the subject (24) I mentioned other types due to penetrating wounds of the venous sinuses, minor infection of the accessory air sinuses, and the type associated with the puerperium which has been described by Martin and Sheehan (25). Mention of these aetiological factors illustrates the importance of this condition in the differential diagnosis from brain abscess. Although the diagnosis may ultimately depend on a ventriculogram or exploratory trephination, there are some clinical observations which may help in the differentiation.

The headache and vomiting are usually less severe than in cases of brain abscess. Indeed, there may be none, and in some cases it is only when the patient develops a squint and diplopia that the fundi are examined and papilloedema is discovered. Thus, whereas headache is more common than papilloedema with brain abscess, the reverse is true of cerebral thrombophlebitis (otitic hydrocephalus). If focal neurological signs are present, they usually develop much more quickly than with a brain abscess, but have not the dramatic suddenness of an embolic lesion. Thus, in cases described in the paper already mentioned (24) the hemiplegia developed over a period of forty-eight hours, and by the end of that time there was complete disruption of all functions of one hemisphere: aphasia, hemiplegia, hemianaesthesia, and hemianopia. In these cases of gross hemisphere dysfunction there is profound stupor or coma, whereas in the type without focal neurological signs (and presumably without thrombosis of cerebral veins) the patient is usually perfectly alert and rational, although he may have intense papilloedema. Thus it would appear that the stupor is more a factor of the gross hemisphere lesion than of increased intracranial pressure.

The spinal fluid pressure is raised, but typically the fluid is normal on analysis, and this is one of the

most important features of the differential diagnosis from brain abscess. There are some cases, however, in which the fluid shows the same changes as in abscess formation - i.e., a slight increase in the protein and cell content. Such cases present difficulties because they are usually the ones with focal neurological abnormalities, but as mentioned above these signs usually develop much more quickly and are more complete than in cases of brain abscess. It should be said, too, that focal epileptic attacks are common in cerebral thrombophlebitis, whereas they are rare in temporal lobe abscess due to mastoiditis. Further, the patient with increased intracranial pressure due to thrombophlebitis is usually less ill than if he had a brain abscess. To summarise, if intracranial symptoms develop during mastoiditis, before or after operation, the common problem is that of differentiating between thrombophlebitis and abscess formation. If the headache is not very severe, the patient not gravely ill, although having papilloedema and an elevated spinal fluid pressure, and if the cerebrospinal fluid is normal on analysis, the lesion is probably thrombophlebitis. If neurological signs (aphasia, hemiplegia, hemianopia, and stupor or coma) develop quickly, say in a period of two or three days, and there are changes in the spinal fluid,

thrombophlebitis is more likely than abscess formation, but in such cases more direct evidence may be necessary. Our practice is to do a ventriculogram if there is any doubt. When the clinical picture is predominantly one of increased intracranial pressure, the ventriculogram is normal, or there may be very slight ventricular dilatation (Fig. 19). This finally excludes the presence of an abscess. But in the cases of thrombophlebitis affecting cerebral veins and causing widespread and profound signs of hemisphere dysfunction the ventricle on the affected side may be collapsed and the opposite ventricle grossly shifted (Fig. 20). These changes are presumably due to massive oedema of the affected hemisphere. When the history can be relied on, it will usually be apparent that the lesion has developed too quickly to be compatible with abscess formation, but there are some cases in which the mode of onset of the neurological signs is not known, and in such cases the only way in which an abscess can be excluded is by trial aspiration in various parts of the hemisphere, e.g., in the frontal, parietal and temporal regions. If pus is not found and no neoplastic tissue encountered, the probability is that the lesion is thrombophlebitis.

Otitic hydrocephalus and cerebral thrombophlebitis are usually benign conditions, and there is a danger of

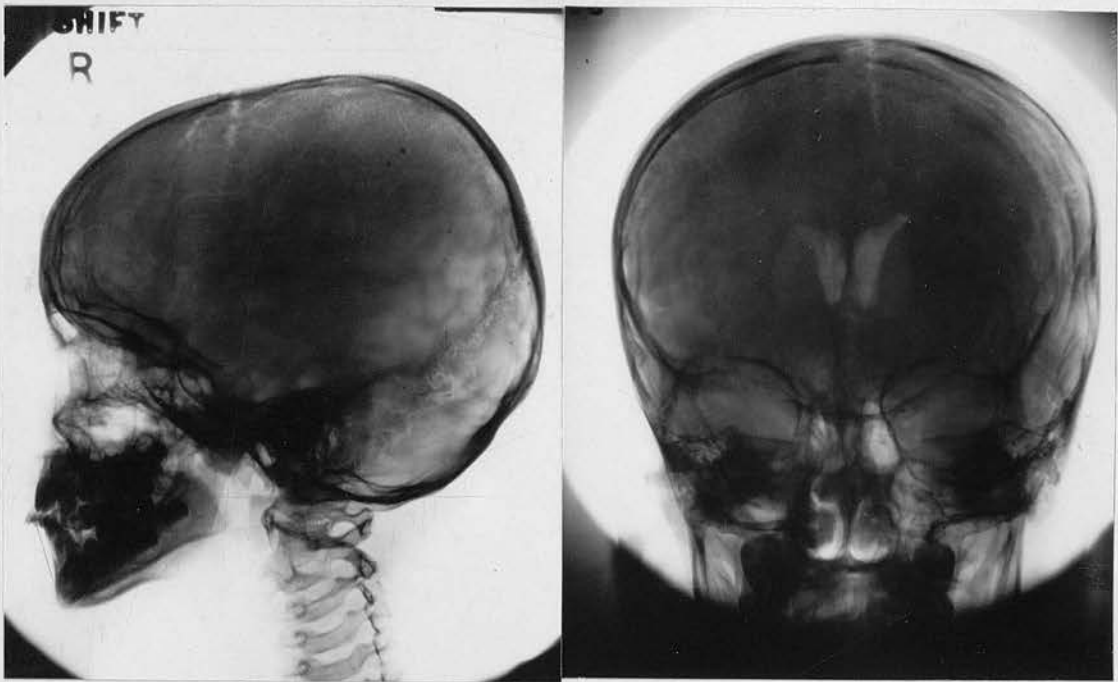


Fig. 19. A case of increased intracranial pressure due to traumatic thrombosis of the right transverse sinus. The arrow in the lateral projection indicates a penetrating wound of the skull over the groove for the sinus, and this was attended by severe venous bleeding. The wound healed without infection but the advent of headache and vomiting led to the suspicion of an intracranial abscess. The ventriculogram was made two months after the injury while the symptoms and signs of increased pressure were at their height. It was normal, and this was taken as strong evidence against the presence of an abscess. The patient subsequently made a complete recovery.



Fig. 20. Ventriculogram in a case of thrombophlebitis of the left hemisphere due to infection of the left maxillary antrum. The patient presented himself during an attack of focal motor epilepsy affecting the right side of the face and the right upper and lower limbs. Within forty eight hours he had developed a complete sensori-motor hemiplegia on the right side, right homonymous hemianopia, and complete aphasia. The ventriculogram was made at this stage : note the absence of filling of the left ventricle and the marked shift to the right of the right ventricle, presumably due to widespread oedema of the left hemisphere. Abscess formation was excluded by multiple aspirations of the left hemisphere. Recovery was uneventful and complete except for the hemianopia which has persisted.

being too ready to accept this diagnosis for unexplained cases of increased intracranial pressure, and for the complications of mastoiditis. Thus, one case in this series was regarded as suffering from otitic hydrocephalus for several weeks before it was discovered that he had a large temporal abscess. The correct diagnosis was made by demonstrating an upper quadrant defect in the opposite field of vision (Fig. 17) and by the fact that every specimen of spinal fluid which was withdrawn by daily lumbar puncture contained an increase in protein and an excess of cells. In less obvious cases ventriculography is the only certain method of establishing the diagnosis.

Other Intracranial Infections.

1. Extradural Abscess usually arises from an infection of the overlying bone, and it may complicate frontal sinusitis, mastoiditis, and infections of the vault of the skull, as in the case described on p. 49. In our experience it is encountered more frequently during operative treatment of the bony infection than because it is sought for primarily. Thus an extradural abscess may be found during a mastoid operation, or when infected bone is removed from the frontal region in a case of frontal sinusitis. If the abscess is large enough and is situated over an eloquent part of the brain, it may cause cerebral compression, as in the case already mentioned in which there was a profound hemiplegia.

The type associated with mastoiditis may **cause** severe local pain around the ear, in the temple, and along the zygomatic arch (Case 11), but otherwise there may be only mild generalised headache and very little local pain, with no neurological abnormalities. The spinal fluid pressure is usually normal, and the fluid is normal on analysis unless the infection has **traversed** the dura to cause a subdural abscess, leptomeningitis, or a brain abscess. Any of these complications may occur if the extradural abscess is not dealt with, and this is one of the reasons for the **energetic** treatment of osteitis of the skull due to whatever cause. Removal of the infected bone and drainage of the extradural abscess are the best insurance against the more serious intradural complications.

2. Subdural Abscess may be a localised collection of pus between the dura and the brain or it may be a more diffuse subdural collection without definite boundaries. The latter type is perhaps better called purulent pachymeningitis, and it is more common than a localised subdural abscess.

Purulent pachymeningitis may be due to blood-borne infections, and the commonest primary sources among our cases have been **chronic** pulmonary and abdominal sepsis. Another source has been chronic pan-sinusitis, especially

of the frontal and ethmoidal air cells in which the infection spreads directly from the adjacent bone. It is an acute and rapidly fatal complication, but the onset may be no more dramatic than occurs with many cases of brain abscess, and the possibility of an abscess usually has to be excluded. There is little in the clinical picture on which the differentiation can be made: the onset and the neurological signs may be similar, but the symptoms and signs of increased intracranial pressure are not as marked in purulent pachymeningitis as in brain abscess. The course of the disease may be of some value in diagnosis too: the patient with purulent pachymeningitis usually deteriorates much more rapidly than is the case with brain abscess. Ordinary X-rays of the skull are generally of no direct assistance, and the cerebrospinal fluid findings are inconstant: we have had some cases of diffuse purulent pachymeningitis with perfectly normal fluid, but in other cases there is an increase in the protein and cell content. The only way in which the diagnosis can be established is by exploratory trephination: in making parietal burr holes for a ventriculogram, subdural pus may be encountered. If not, and the ventriculogram does not disclose a lesion adequate to explain the clinical picture, other burr holes may have to be made. Thus in 2 cases of purulent pachymeningitis due to chronic sinusitis the subdural space

and cortex in the parietal region appeared to be normal, and there was no specific ventricular deformity. A burr hole in the frontal region disclosed a diffuse collection of pus in the subdural space, which at the autopsy was found to be largely confined to the superolateral, medial, and inferior surfaces of the frontal lobe.

Localised subdural abscess occasionally occurs as a complication of mastoiditis. A shallow pool of pus accumulates between the dura and the lateral surface of the temporal lobe, and this process is usually associated with necrosis of the dura and of the cortex forming the inner wall of the abscess. There may be focal neurological abnormalities (e.g., aphasia, hemiparesis) which lead to the suspicion of an intracerebral abscess, and as with the more diffuse suppuration described above there may be nothing in the clinical picture or in the examination of the cerebrospinal fluid on which the differentiation can be made with certainty. Exploratory trephination will usually provide the diagnosis: a burr hole in the squamous temporal bone just above the external auditory meatus (Fig.25 p.127) will allow an escape of pus as soon as the dura is incised.

3. There is generally not **much** difficulty in the diagnosis of leptomeningitis because the spinal fluid findings are characteristic. Clinically, the signs of meningeal

irritation predominate over those due to increased intracranial pressure and to focal lesions of the brain. But a difficulty may arise in detecting a brain abscess which exists along with or causes meningitis. Thus Fig. 21 relates to a small inferior frontal abscess which ruptured into the anterior horn of the ventricle to produce a clinical picture of meningitis without any focal neurological disorders. In the presence of widespread meningitis, diagnosis and treatment of an abscess are of less immediate importance than dealing with the meningitis, but any unexplained residuum of signs or symptoms **should** raise suspicion of a brain abscess, and the most certain diagnostic procedure is ventriculography. The ventricular system commonly becomes a little dilated during an attack of meningitis, but the dilatation is symmetrical and uniform, and there should be no displacement or specific deformities unless there is a localised collection of pus.

The mild meningitis which may occur during the incubation or treatment of a brain abscess has already been mentioned (p. 36). In these cases there is generally no difficulty in detecting the brain abscess because the meningeal signs and the changes in the spinal fluid are generally too slight to account for all of the symptoms; and the symptoms and signs of the abscess

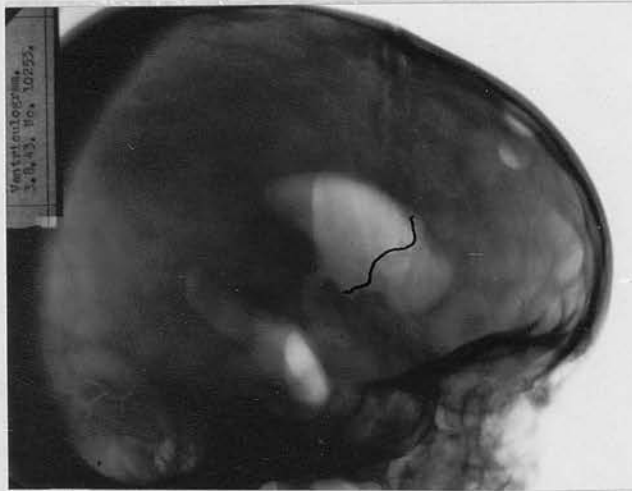
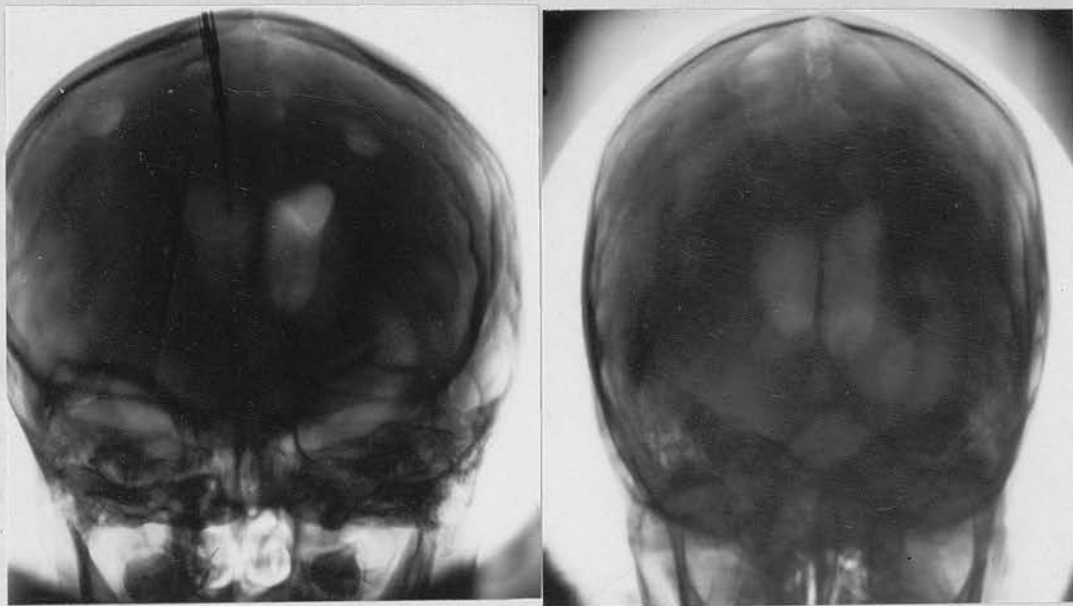
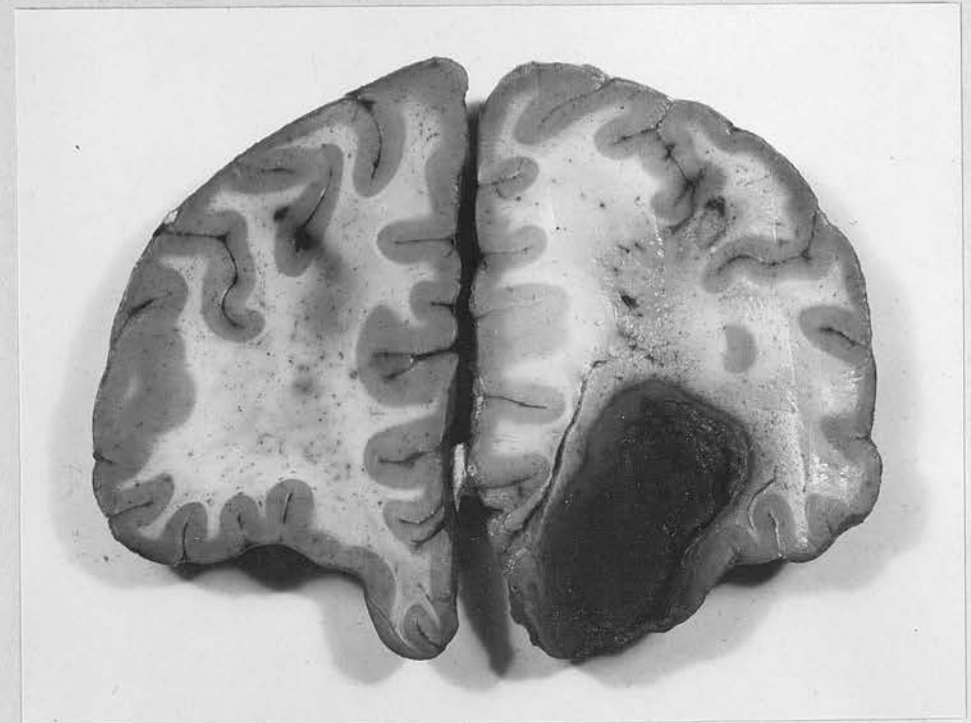


Fig. 21 . Ventriculograms of a case of meningitis due to leakage of a right inferior frontal abscess into the ventricle. Note the shortening and indentation of the right frontal horn due to the abscess seen in the photographs at the right (in which the sections of the brain are viewed from behind). Also note the absence of lateral displacement and the generalised ventricular dilatation due to meningitis. Compare these appearances with Fig. 15 in which the right frontal abscess was not accompanied by meningitis.

The abscess in this case probably resulted from a chronic nasal infection due to the impaction of a foreign body in the nose. It caused no symptoms until a trivial head injury was followed after twenty four hours by the abrupt onset of meningitis without any localising signs.



persist when the meningeal reaction has subsided.

4. Tuberculous Meningitis. The clinical picture and course of tuberculous meningitis are so inconstant that it frequently has to be considered in the differential diagnosis. Thus many patients with tuberculous meningitis have chronic mastoid infections, or other likely sources of a brain abscess, and the onset of intracranial symptoms and signs may lead to the suspicion of a pyogenic infection before tuberculous meningitis is suspected. Examination of the cerebrospinal fluid is the most important diagnostic procedure: demonstration of the tubercle bacillus leaves no doubt, but otherwise the most characteristic feature of a tuberculous infection is progressive diminution of the chloride content. It may not be possible to make the diagnosis on a single examination of the fluid, but repeated examinations should show a progressive diminution in the chloride content. It should be said, however, that a low chloride content is occasionally met with in pyogenic infections, and an instance has been cited (p. 64) in which a persistently low chloride content led to the mistaken diagnosis of tuberculous meningitis.

In cases suspected of tuberculous infection, the fundi should be examined carefully for tubercles in the choroid, as they are by no means rare, and we have found

them before it was possible to establish the diagnosis on the spinal fluid findings. The chest should be X-rayed, and other common sites of a primary tuberculous infection carefully investigated. If the evidence is doubtful it may be necessary to exclude the presence of an abscess by doing a ventriculogram, and this usually shows a slight ventricular dilatation without displacement or deformity (Fig. 22).

5. Petrositis. In some cases of mastoiditis the infection extends beyond the pneumatised area to the apex of the petrous bone. The patient usually complains of intense pain around the ear and in the temple, and it may be so severe that he is unable to differentiate it from a headache. Ipsilateral sixth nerve palsy is common, and with the pain may be mistaken for a sign of increased intracranial pressure. There are usually no other focal neurological abnormalities, but in some cases there may be trigeminal pain and sensory impairment. These signs are due to local effects on the fifth and sixth nerves owing to their immediate proximity to the infected bone, and the spinal fluid may show the changes to be expected from localised inflammation of the meninges - i.e., a slight increase in the protein and cell content. The pressure is usually not elevated, however, and there are no changes in the fundi unless,



Fig. 22. Ventriculogram in a case of tuberculous meningitis.

Note the symmetrical generalised dilatation of the ventricles which in this case had occurred ten days after the onset of symptoms.

as sometimes happens, the infection spreads to the cavernous sinus, when there may be congestion or frank papilloedema, together with multiple ocular palsies. X-rays may show evidence of focal destruction of the petrous bone, and small sequestra may be seen. Unless the changes are definite the exclusion of an intracranial abscess may depend on a ventriculogram. Fig. 23 shows the slight degree of ventricular dilatation seen in a case of petrositis. The dilatation is probably due to a mild chronic meningitis, and in this case it excluded the possibility of a temporal abscess.

6. Arterial Hypertension. We have encountered several patients who have been referred as suffering from a brain abscess because of their having chronic mastoid or sinus disease, severe headache, and changes in the fundi suggesting increased intracranial pressure. That these intracranial symptoms were due, not to an abscess, but to high blood pressure was suggested by the sphygmomanometer readings, which had usually not been taken by the doctor who diagnosed cerebral abscess. A person with hypertension may also have an abscess, of course, and it may be difficult to differentiate the headache of arterial hypertension from that due to increased intracranial pressure: both may be very severe, worse in the early morning on waking, and associated

with vomiting. It may also be difficult to differentiate the changes in the fundi due to arterial disease from those due to increased intracranial pressure, even for an experienced ophthalmologist. In this type of arterial hypertension there are usually no focal neurological abnormalities, but signs may appear as the result of thrombosis or haemorrhage. The spinal fluid pressure may be elevated, but it is always normal on analysis. In some cases differentiation from an abscess can only be made with certainty by ventriculography. In arterial hypertension the ventricles are normal or they may be slightly dilated if there is some atrophy of the brain due to impaired blood supply (Fig. 24).

7. Psychoneurosis. Ear, nose and throat surgeons see a good many patients with chronic mastoid or sinus disease who complain of severe headache out of all proportion to the clinical and radiographic abnormalities. Indeed, in many of them the headache persists after the infection has completely cleared up. The constant complaint of headache sooner or later raises the suspicion of an abscess of the brain, either to the patient or to the general practitioner or specialist who has to bear the brunt of the complaint. The patient's description of the headache is usually unlike that of any organic affection, and there are no neurological abnormalities.

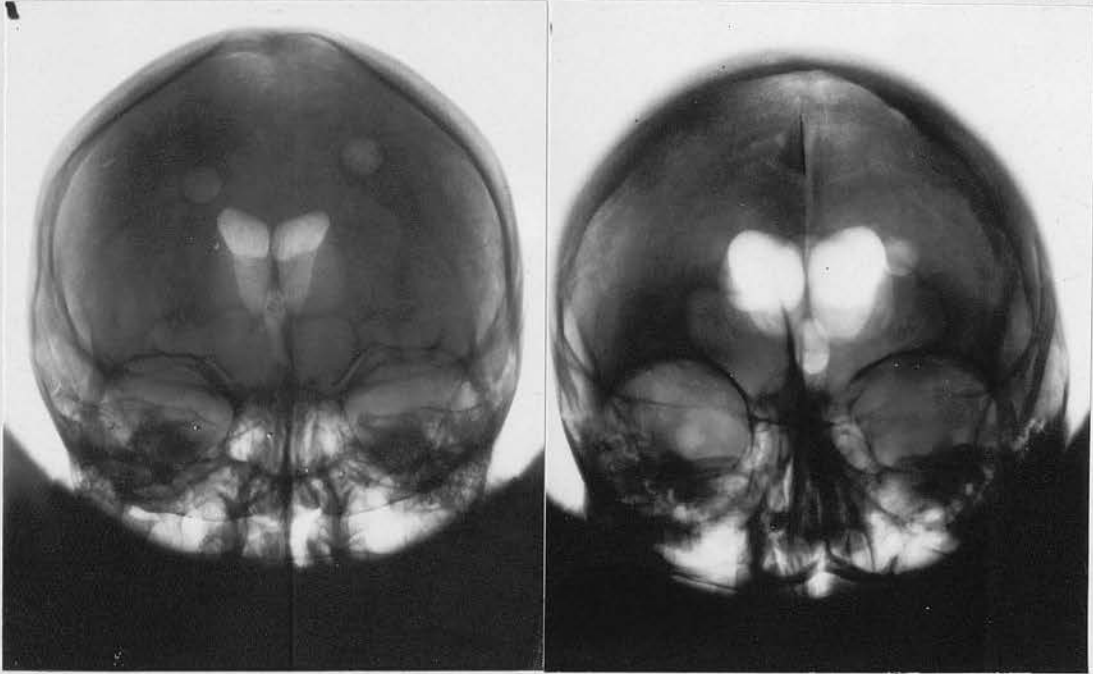


Fig. 24. Ventriculograms in two cases of arterial hypertension with intracranial symptoms. The plates show slight and moderate degrees of ventricular dilatation without deformity or displacement. Such appearances are due to diffuse atrophy consequent on impaired blood supply.

The spinal fluid pressure is normal, and the fluid is normal on analysis. If this evidence is not impressive enough and if the patient or his doctors want absolute assurance that there is no abscess, a cisternal encephalogram should be done. In some cases this evidence of normality may be enough to reassure the patient and to cause some improvement. There are others in which nothing helps and drug addiction may occur. These are psychiatric problems, and the assistance of a competent psychiatrist should be enlisted when it is certain that there are no organic abnormalities.

VII. Treatment.

In this section I have analysed the methods employed in the treatment of the first forty cases of the series, as sufficient time has elapsed to enable an accurate assessment of the late results to be made and largely to exclude the possibility of recurrences. The ten cases which are not included comprise two cerebellar abscesses and one bronchogenic abscess which proved fatal, Case 20, the occipital abscess due to gunshot wound mentioned on p. 22, four cases which have been discharged as cured within the last few months, and one case which is convalescent at present.

TABLE II. RESULTS OF TREATMENT.

	No. of Cases	Deaths
I. <u>Metastatic.</u>	13	6
A. <u>Thoracogenic</u> (lung abscesses, bronchiectasis 5; infective endocarditis 2)	(7)	(7)
B. <u>Other Sources</u> (boils, carbuncles, etc.)	(6)	(1)
II. <u>Mastoid Disease.</u>	15	8
A. <u>Cerebral</u>	(10)	(4)
B. <u>Cerebellar</u>	(5)	(4)
III. <u>Frontal Sinus Disease.</u>	6	0
IV. <u>Penetrating Wounds, Infection of Scalp and Skull.</u>	6	0
TOTAL	40	16

In 7 of the 40 cases in this series the intracranial infection was a terminal complication of diseases for which at present we have no cure, i.e., bronchiectasis and infective endocarditis. One case was undiagnosed and untreated; 3 cases were in extremis when they were first seen. If these are excluded, there are left 29 cases in which it was felt that treatment had a chance

of success. Of these, 5 died, a mortality rate of 17 per cent. For comparison with this series, I reviewed the records of 40 consecutive cases of brain abscess in the London Hospital (1933-37). They were roughly comparable with the Oxford series as to origin. Applying the same correction to this as to the Oxford series (i.e., excluding the bronchogenic abscesses, the undiagnosed and untreated cases, and those which from the records appear to have been in extremis when first seen) there were left 29 cases (as in the Oxford series), of which 19 died, a mortality rate of 65 per cent.

One of the most important considerations in comparing these results (see footnote) is that all of the Oxford cases had the advantage of chemotherapy, whereas only one of the London Hospital cases was so treated. It might be thought that this is a sufficient explanation for the difference, but most surgeons would agree that the chemotherapeutic agents at present available, although powerful allies, cannot deal with a brain abscess alone. It will be seen that the methods of surgical treatment

A number of factors worked to the advantage of the Oxford series where the cases were all under observation in a single ward, with the co-operation of trained residents who could recognise indications for urgent treatment. The X-ray Department of the Radcliffe Infirmary was always at our disposal, and there was always an operating theatre available for emergency procedures. These factors contribute largely to the results and should be considered when planning for the treatment of these cases.

differed considerably (Tables III and IV), and it is with what appears to be a significant development in the treatment of brain abscess that this section is chiefly concerned.

It has been shown that the two factors which, operating singly or together, cause death in cases of brain abscess are (1) dissemination of the infection throughout the nervous system, and (2) increased intracranial pressure resulting partly from the bulk of the abscess and partly from cerebral oedema. **Treatment** must thus be concerned with both of these factors.

Most of the literature on treatment is concerned with various drainage procedures. Macewen's (1) practice was to incise the dura over the abscess, put in sinus forceps or a cannula, and allow the pus to well out. In some cases the abscess cavity was irrigated with an antiseptic solution (weak carbolic acid). Continued drainage was provided for by a decalcified chicken bone which acted as an absorbable tube. The reports indicate that there was rarely any profuse discharge and the dressing was usually not changed for 3 weeks, at the end of which time the chicken bone had been absorbed and the wound was covered by a layer of granulation tissue.

This method can be described briefly as one of incision and drainage. It has been and still is

extensively employed, with the substitution of a rubber tube for the chicken bone. It is the simplest procedure known for the treatment of brain abscess, and the fact that no one has been able to equal Macewen's results calls for some explanation. A very careful selection must have been exercised, as his 25 cases referred to above were collected over a 12-year period (1881-1893), and yet the standards for selection are not very obvious in the reports: some cases which appeared to be in extremis were operated on, and others which at present would be considered operable were left to die without operation several days after admission to hospital. The most illuminating point, however, is the size of the abscesses: they were for the most part very large, containing two or three ounces of pus, and he speaks of disappointment in one case at finding only half an ounce of pus in a cerebellar abscess. That they attained this size without causing death indicates that **they** were fairly chronic, and, as usual with such abscesses, they had a single locus. It has been the experience of many surgeons since Macewen's time that these large unilocular abscesses are favourable for drainage, and the failure to reproduce his results lies in the attempt to apply this as a general principle of treatment in all cases. A number of reasons for the failure of this

method will be obvious from the brief considerations of the pathology set out above. There is difficulty in getting a drainage tube to rest efficiently in a small acute abscess: the surrounding oedema squeezes the walls of the cavity together as soon as the pus is released, and the wall being thin it is usually impossible to say whether the end of the tube is actually in the cavity or has transfixed it. If the latter, another abscess will probably develop around the end of the tube which has become contaminated by passage through the infected cavity. Drainage by a tube is generally ineffective in multilocular abscesses: while there may be some drainage from a known loculus, another untouched loculus may be ripening for rupture into the ventricle or subarachnoid system. Or the undrained loculus may be so large as to cause a fatal increase in intracranial pressure, despite a slight discharge from the drainage tube.

Dissatisfaction with tube drainage led to various methods of "open drainage". One of the earlier procedures was marsupialisation of the abscess by making a craniotomy over the lesion, exposing the surface of the capsule, stitching it to the dura or pericranium, and then opening the cavity and allowing it to drain externally (Horrax, 26). Another method advocated by King (27) and practised with great success by him is

to make a relatively small craniotomy, remove the dura and cortex over the abscess, open into the cavity and evacuate the pus, then to pack the cavity lightly with gauze soaked in an antiseptic solution. The surrounding oedema extrudes the abscess cavity through the opening in the skull, the size of the fungus being controlled by frequent lumbar punctures.

The advantage of both of these methods is that they allow a certain decompression effect by virtue of the craniotomy and that they permit at least a limited inspection of the abscess itself. Neither method deals satisfactorily with the problem of multiple loculi, however; it is possible that one loculus may be marsupialised or extruded as a fungus and a deeper loculus remains undetected. It should be said too that marsupialisation is only possible in those cases where the abscess has attained a fairly thick capsule, i.e., thick enough to hold stitches. King's method is generally more applicable in the acute stage before a firm capsule is formed. Both methods entail frequent dressings, a relatively long hospitalisation, and unremitting personal care of the surgeon in charge. These are not serious objections, but any experience of either method is sufficient to show that improvements should be welcome.

More recent methods of treatment have aimed at the

extirpation of the whole of the infected mass. There is nothing new about the excision of a chronic encapsulated abscess: most neurosurgeons have had the experience of removing a thick-walled abscess embedded in the brain, sometimes in the belief that it is a neoplasm. But this method was never applied as a principle of treatment until Vincent (28) showed that a subacute abscess may be tided over to the chronic encapsulated stage by repeated aspiration and bony decompression. Briefly, his method is to localise the abscess accurately (by ventriculography, if necessary) and to reflect an osteoplastic flap over it. The abscess is then aspirated through the unopened dura, the flap is replaced, and the wound closed. As more pus forms further aspirations are done through a suitably-placed trephine hole in the flap. The aspirations are repeated as often as necessary until the abscess has attained a thick wall, and then the flap is re-elevated and the lesion is dissected out intact, much as a solid neoplasm.

We have found that the bony decompression is often unnecessary because the size and tension of the abscess can be controlled by repeated aspirations, governed by clinical observations, and radiological evidence obtained by visualising the abscess with thorotrast (v.i.). By so doing we have minimised the risk of infection of the bone flap and have reduced by one stage the major operative procedures.

The virtue of extirpation lies in the fact that once an abscess has been removed from the brain there is none of the possibility of recurrence which attends less radical methods in which the wall of the abscess, or some part of it, is left as a fibrous scar in the brain. Many months or years may pass before some dormant organisms in this scar recover their vitality and cause a recurrent infection (see Test of Cure).

The following Table indicates the methods of treatment employed in this series.

TABLE III.

Method	No. of Cases	Recovered	Died	Remarks
I. Simple drainage by tube	1	-	1	This was an acute abscess in the right parietal lobe following empyema thoracis which had resolved.
II. Decompression (without aspiration)	2	-	2	1 was an abscess in the vermis of the cerebellum which has been mentioned in the text (p.); the other was a small chronic cerebellar abscess which was not located at the time of the decompression.
III. Marsupialisation	(2)	(2)	-	Both of these cases required extirpation ultimately and are entered in Group VII.
IV. Fungus method (King)	6	-	6	3 of these were cases of multiple bronchogenic abscesses; the other 3 were temporal abscesses consequent on mastoid disease in which the operation was undertaken in extremis.
V. Primary extirpation	7	5	2	3 of the cases which recovered were chronic abscesses which were not recognised until operation. The 2 fatal cases were both abscesses due to mastoid disease, 1 in the temporal lobe, 1 in the cerebellum. They were extirpated before they were "ripe".
VI. Aspiration + decompression	6	5	1*	In these cases the aspiration and decompression were undertaken as preliminary to extirpation, but the abscesses resolved without further treatment being necessary (see Test of Cure).
VII. Aspiration, decompression & subsequent extirpation	16	14	2	1 of the fatal cases was a multilocular cerebellar abscess following mastoid disease; the other was a case of multiple bronchogenic abscesses in both hemispheres which has been mentioned in the text (Case 1).
Undiagnosed &/or untreated	2	-	2	1 was an abscess due to infective endocarditis; the other a metastatic cerebellar abscess following boils in which a diagnosis of tuberculous meningitis was made.
TOTAL	40 ⁺	24	16	

* Infective endocarditis.

⁺ Of the last 10 cases in the series 5 recovered after extirpation and will subsequently be entered in Group VII. One of the fatal cerebellar abscesses was also treated by aspiration, decompression and extirpation and would be entered in this group. The other fatal cerebellar abscess was treated by aspiration and decompression and would fall into Group VI. In both of these the cause of death was diffuse meningitis. In Case 20 and the case reported on p.22 the

Table III is a brief historical summary of the progress of treatment. It is obviously of no value in comparing the efficacy of the various methods because they were not employed impartially, either as regards numbers or the type of abscess. Thus, that all of the cases treated by the fungus method died does not decry that method because they were all desperate cases anyway; indeed, it may be worth a more serious study, as King's results (17 cases, with 14 recoveries) challenge any which have been published. That only one case was treated by simple drainage was due to a considerable and unsatisfactory experience of that method in earlier cases: an analysis of the methods of treatment employed in the London Hospital series summarises this experience.

diagnosis was missed. The case of bronchogenic abscess was one of multiple abscesses throughout the brain and was treated by aspiration.

Since this series was closed there have been three acute temporal abscesses, all treated by aspiration and extirpation, and all have recovered. Thus there are at present 25 cases in Group VII with 3 deaths, a mortality rate for this group of 12 per cent.

TABLE IV. METHODS OF TREATMENT EMPLOYED IN LONDON HOSPITAL
SERIES (TABLE II).

	Method	No. of Cases	Recovered	Died
I.	Drainage by tube	17	4	13
II.	Decompression	1	-	1
III.	Aspiration	4	-	4
IV.	Decompression - aspiration	5	-	5
V.	Marsupialisation	3	1	2
VI.	Excision	5	5	-
	Undiagnosed &/or untreated	5	-	5
	TOTAL	40	10	30

The present method of treatment aims at the eventual extirpation of the abscess and the evolution of this method is apparent from a study of Tables III and IV and from consideration of the pathology set out above. The extirpation of an abscess demands that it has a sufficiently thick wall to permit dissection from the surrounding brain, preferably without any accidental rupture. The formation of such a capsule takes from six to ten weeks from the onset of the cerebral infection, as judged from the few cases in which it has been possible to assess the age of the abscess. During this time in untreated cases one of several things may happen: the abscess may leak or rupture into the ventricle or subarachnoid space, causing a fatal meningitis; the patient may die from increased intracranial pressure; or he may become blind from secondary optic atrophy.

The primary concern of treatment, then, is to save the patient's life and eyesight until the abscess can be excised. These indications can generally be met by repeated aspirations, although in some cases additional decompression by a craniotomy may be necessary. A significant advance in Vincent's method was the decompression effect afforded by the osteoplastic flap: the efficacy of such a decompression had been observed in many cases of increased pressure due to neoplasms. The

dura stretches and allows the brain to expand slowly, sometimes too slowly to meet the needs of an acute abscess, and in these cases the dura may have to be opened (v.i.).

1. The Time for Treatment. In general, it is wise to defer treatment until the abscess has attained some size. Thus it is often possible to diagnose and localise an abscess in its very early stages before it has produced any alarming symptoms, e.g., of increased intracranial pressure. The common indications for beginning treatment are (1) severe and intractable headache; (2) the onset of drowsiness and slowing of the pulse rate. Some cases have been kept under observation deliberately for 2-3 weeks before these indications were manifest, and it is perhaps a weakness of this method that no more precise rule can be given as to the exact time for intervention. The time spent in waiting can be employed usefully in general sulphonamide therapy (against the risk of meningitis), in dealing with the primary infection (e.g., of the mastoid or lung) if it still persists, and in building up the patient's general resistance for what may be a long illness by feeding, cleaning up a dirty mouth, etc.

2. Localisation. When the time for intervention arrives, it is essential to know the precise location of the abscess. It has been shown that the majority of temporal

lobe abscesses following mastoid disease are in a fairly constant position, and a burr hole in the squamous temporal bone immediately above the external auditory meatus is usually adequate for the aspiration (Fig. 25). Similarly, with unilateral frontal sinusitis producing definite signs of a related frontal lobe abscess, a burr hole in the vicinity of the hair line is suitable (Fig. 25). Sometimes a difficulty arises in cases of bilateral mastoid or frontal sinus disease producing indefinite signs, and in these cases, as in all where there is any doubt, more precise information is necessary, and this usually means ventriculography. In many acute cases the ventricle in the affected hemisphere cannot be found, and air injected into the opposite ventricle fills only that cavity. In these cases it is presumably extensive oedema of the affected hemisphere which obliterates the ventricle, and the ventriculogram only gives information as to lateralisation. If there is a history of frontal sinus or middle ear infection, the presumption will be that the pus is in the frontal or temporal lobe, and the exploratory aspirations directed accordingly. If both ventricles are filled, the ventriculogram will usually show deformity and displacement sufficient to localise the lesion (Figs. 15 and 21, p.109). In the case of cerebellar abscess, there is usually a moderate



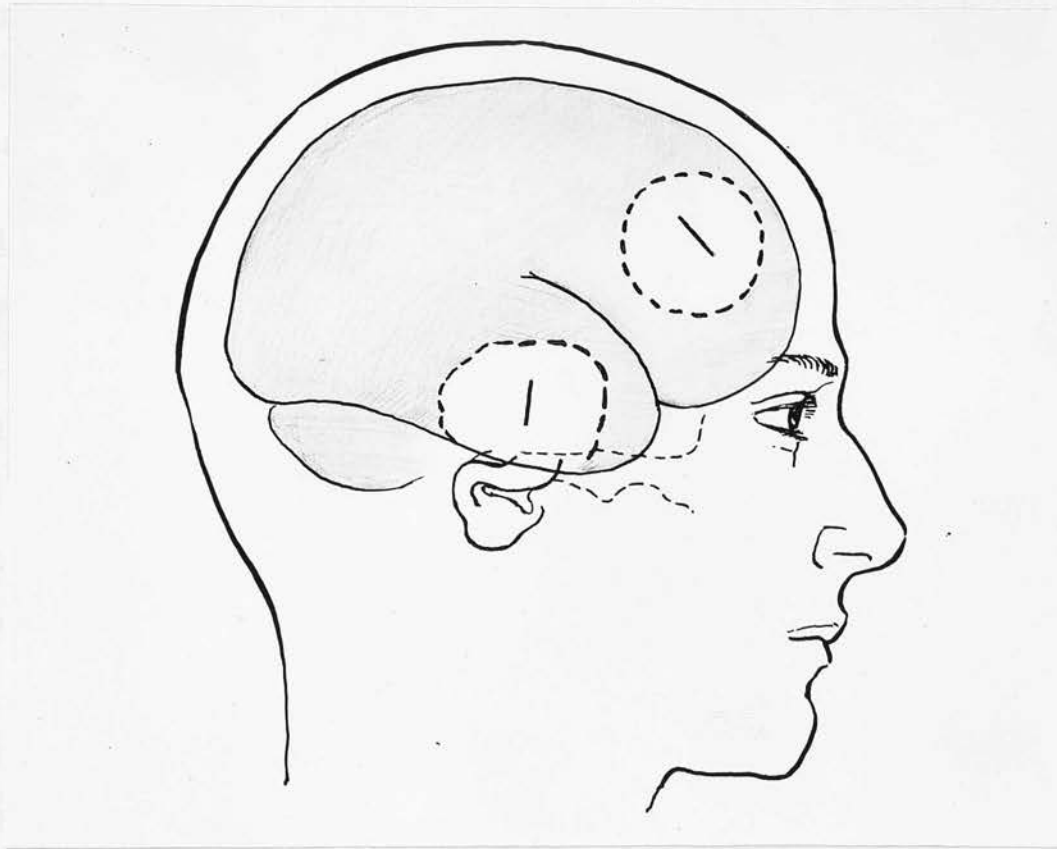
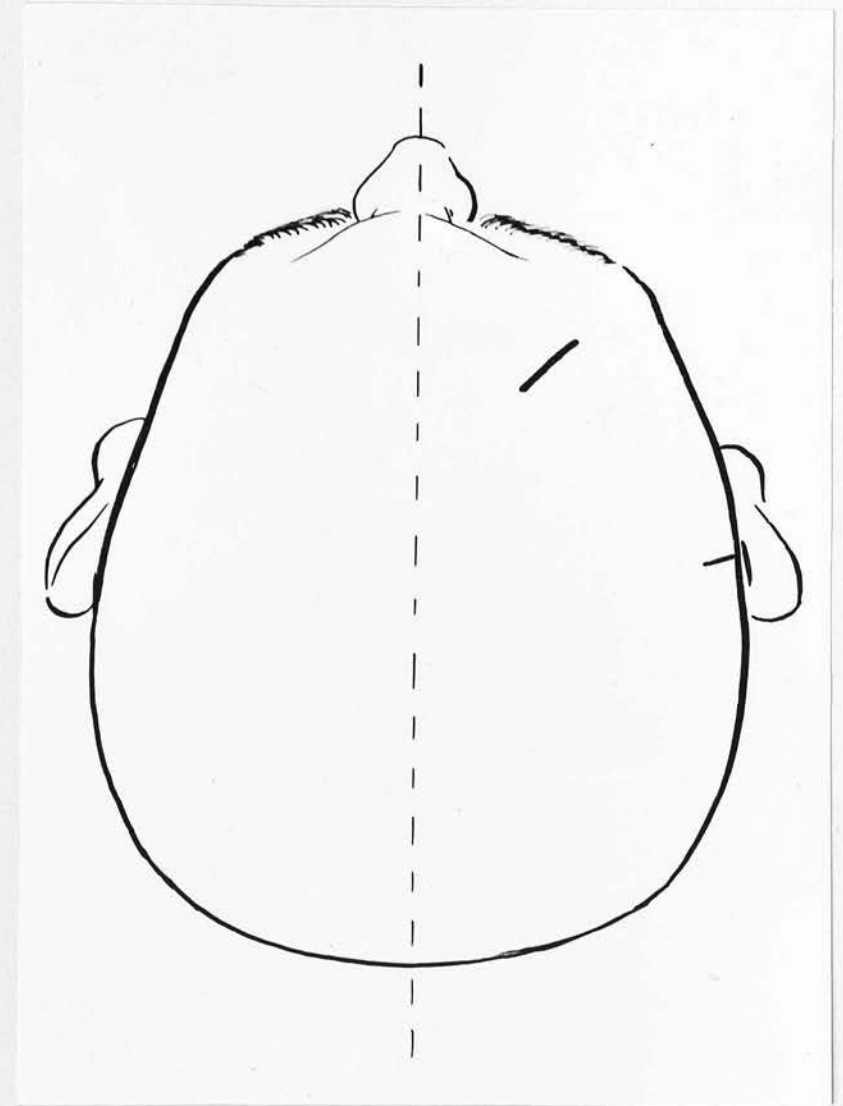


Fig. 25. Incisions used for aspiration of temporal and frontal abscesses. Such incisions are radii of the standard scalp incisions used for the reflection of osteoplastic flaps in the later stages of treatment, and thus do not interfere with the blood supply of those flaps.



dilatation of the lateral and third ventricles which may be sufficient for diagnostic purposes (Fig. 40 , p.212), but in some cases lateral displacement of the aqueduct and fourth ventricle can be visualised.

3. Aspiration. Having knowledge of the site of the abscess a burr hole is made over it, using local anaesthesia. In children and refractory adults sedation by chloral or light pentothal anaesthesia can be employed safely. The dura is incised sufficiently to admit a blunt brain needle which is introduced towards the abscess. The blunt needle enables one to feel whether or not there is any resistance, as of a capsule. In the early abscesses there is usually no definite resistance, but there may be a sudden "give" when the needle enters the cavity. The needle is then held firmly in position and the stilette is withdrawn. If the tension in the abscess is high, pus will start welling out of the needle. Sometimes it is so thick that it has to be aspirated by gentle suction with a rubber-bulb or piston syringe; in any case, this is a convenient method of collecting the pus and preventing contamination of the wound. There may be only 8-10 c.c., or there may be 80 c.c. It is wise not to move the needle in a search for more pus, but to see what is the effect of the initial aspiration. The reason for this is that the point of

the needle is contaminated and it may "plant" another focus of infection in the brain around the abscess.

If there is no feeling of the exploring needle having entered a cavity the needle should be passed in to a depth of 3-4 centimetres from the dura, the stylette withdrawn, and gentle suction applied with a rubber-bulb syringe. While maintaining the suction the needle can be moved backwards and forwards in its own axis until the abscess cavity is located. Having found the pus, it is important to steady the needle during the aspiration to keep from "losing" the cavity.

4. Instillation of Thorotrast. Having aspirated as much pus as possible the next step is to instil 2 c.c. of thorotrast into the cavity. The stylette is then replaced and the needle carefully withdrawn. The wound is irrigated with an antiseptic solution (proflavine or azochloramide) and closed without drainage. Healing takes place by primary union.

The instillation of thorotrast was suggested by Kahn (18) and it represents a considerable technical advance. The original purpose was to render the abscess cavity opaque to X-rays and thus enable its site, extent, and variations in size to be determined. This is valuable information and no risks are entailed. Not only is the thorotrast innocuous, but there is some

clinical and pathological evidence to suggest that it actively promotes capsule-formation. In abscesses produced experimentally, Falconer, Russell and Macfarlan (11) found that the thorotrast granules seemed to excite a microglial and reticular fibre reaction round the deeper part of the abscess, i.e., the part which is slowest in acquiring a robust capsule by natural processes.

The effect of the aspiration is usually apparent at once. Depending on the amount of pus removed, the dura becomes slack and the brain falls away from the dural opening. Headache is relieved, the patient becomes more alert, the pulse rate increases slightly, and the neurological signs (aphasia, hemiplegia, hemianopia) become less marked. The general effects are attributable to the relief of increased intracranial pressure, and this can be seen manometrically if a lumbar puncture needle is left in place during the aspiration: in the case of a frontal lobe abscess, we have found the pressure in the lumbar theca to be over 300 mm. water before the aspiration and after the removal of 15 c.c. of pus the lumbar pressure had dropped to 90 mm. The neurological improvement is due to the relief of local pressure in eloquent parts of the cortex or fibre tracts and is comparable to the improvement which may follow aspiration of a cystic neoplasm.

The improvement is usually temporary, and as more pus forms symptoms and signs of increased intracranial pressure again become manifest, and again it is drowsiness which indicates that a further aspiration is necessary. This may be within 48 hours of the initial aspiration or it may not be for ten days to a fortnight afterwards. In the meantime, X-rays of the skull will show the abscess delineated by thorotrast (pyogram) and the increase in size can be followed by photographs taken at regular intervals.

5. Subsequent Aspirations are conveniently done through a stout lumbar puncture needle of the Greenfield pattern. A novocain weal is raised to one side of the scalp incision over the burr hole and the sharp needle introduced into the abscess. This can be done on the X-ray with fluoroscopic control, but generally the pyograms have given sufficient information to enable the needle to be placed accurately in the cavity without "seeking". (With a sharp needle passed through the scalp it is generally not possible to feel the capsule until it has attained great toughness, and this is a sign that the time has come when it can be extirpated.) The stilette is withdrawn, a piston syringe applied, and the pus aspirated gently, care being taken not to move the needle. When no more pus is obtained, the stilette is replaced and

the needle withdrawn. It is not necessary to instil more thorotrast, as generally enough will have been fixed in the wall to preserve radio-opacity.

Again the effect is observed. Usually there is improvement as after the first aspiration, and the patient is left until a recurrence of symptoms and signs demands further aspirations which may need to be repeated six or eight times over a period of as many weeks. An interesting sequel of repeated aspirations has been observed on several occasions: the patient may lose his symptoms and signs and one might feel that the abscess had been "cured", except that examination of the fundi reveals progressive papilloedema. Indeed, one case treated during the acute stage in another hospital became completely blind from secondary optic atrophy without any symptoms of increased intracranial pressure at all. These cases all have a high spinal fluid pressure and changes in the cerebrospinal fluid (see Test of Cure), but they may be missed if these simple investigations are not pursued and if a careful watch is not kept on the state of the fundi and visual acuity. One case in this series (Case 10) was apparently cured by repeated aspirations, and there are reports of many similar cases in the literature. Schreiber (29) has recently advocated cannulation as a method of treatment for cerebellar abscesses.

6. Decompression. There are some cases in which aspiration of all the pus which can be obtained brings about little, if any, relief from the symptoms and signs of increased pressure. These are generally acute abscesses of small size (8-10 c.c.) and it is oedema which contributes most to the high pressure. Another possibility which cannot be excluded safely is that the needle has been in only one small loculus of a multilocular abscess. Nevertheless, it is unwise to pass the needle in various directions seeking a larger loculus, as the oedematous white matter around an abscess is fertile soil for the implantation of a new focus of infection. If oedema is the chief factor, some relief may be obtained by dehydration, e.g., the intravenous injection of hypertonic solutions of saline, glucose, or sucrose, or by giving rectal infusions of magnesium sulphate in saturated solution. The experience in this series has been that such measures have rarely been of more than transient benefit, and a decompression becomes necessary.

The decompression should be planned so that it will suffice for the ultimate excision of the abscess, and the operation usually amounts to the reflection of an osteoplastic flap designed for that purpose. When the bone flap is elevated, another one or two attempts may be made to aspirate the abscess through the unopened

dura, as in Vincent's method. If this is successful and the aspiration slackens the dura, the bone flap should be replaced and the wound closed. If no pus is found and the dura is not excessively tight, it may be worth while replacing the bone flap and watching the effect. That this allows some decompression may be shown from improvement in the clinical condition of the patient, and if some weeks elapse before the flap is re-elevated for extirpation of the abscess, the intact dura may be found to be herniated to the level of the surrounding skull.

If the brain is very tight and no relief has been obtained from the bony decompression, the dura should be opened. Before it can be opened safely the pressure should be lowered by the intravenous injection of hypertonic saline, or by tapping the opposite ventricle. If these precautions are not taken, the brain will herniate rapidly through the dural opening, vessels will rupture, and there may be great difficulty in getting the wound closed. If this happens, there will be further swelling of the damaged brain, the effect of the decompression is lost, and the patient may die either from increased pressure or rupture of the abscess due to the sudden alteration in pressure around it. If, however, the dura can be opened widely and the bone flap replaced quickly

without cortical laceration, the decompression will probably be effective. To prevent adhesion of the cortex to the under-surface of the bone flap, small strips of gutta percha tissue may be laid over the exposed cortex so as to overlap the margins of the dural opening by 1.5 - 2 cm. on each side. This is usually adequate to cover the additional cortex likely to be extruded between the dural margins in the interval between the decompression and the subsequent re-elevation of the flap. The gutta percha seems to excite no untoward reaction and it can be lifted out when the flap is re-elevated leaving the exposed cortex intact.

Once the dura has been opened the decompression effect can be augmented by frequent lumbar punctures. This procedure is now devoid of risk, and amounts of 10-15 c.c. of cerebrospinal fluid can be removed once or twice a day to reduce the pressure to normal (120-140 mm. water) or until the decompression feels slack. The use of dehydrating agents may be of value too, particularly in the first few days after operation when it is important to prevent undue tension on the suture line.

If these measures are successful, there will be a gradual clinical improvement: stupor and headache lessen, the appetite returns (and may become voracious to make up for lack of nourishment during the stupor phases)

incontinence ceases, and the focal neurological signs improve, although less dramatically than after aspiration of a large volume of pus. With this improvement the decompression will be found to be fairly tense, and the bone flap will begin to "ride", signs of the need for and efficacy of the decompression. The spinal fluid pressure is raised (its "edge" taken off by the decompression) and the fluid will contain an excess of protein and a slight lymphocytic pleocytosis. Serial pyograms will show that the abscess is increasing in size very gradually, but it is probably being displaced slightly into the decompression and rendered more accessible for removal. (See Case 1, page 160).

While these things are happening, the patient usually is regarded as having passed the critical phase of his illness and he drops out of the daily - or sometimes hourly - communiques. The abscess is developing a wall, and the management now is largely a matter of waiting until the wall is thick enough to allow extirpation of the intact mass. There are two ways of telling when this has happened: (1) by the passage of time; if conditions will allow eight or ten weeks to elapse after the initial aspiration it can be assumed generally that the abscess wall is tough enough to be extirpated; (2) by feeling with a blunt needle: this means reopening the burr hole used for the earlier aspirations, and if the

needle does not pierce the wall easily the wall is probably thick enough for removal. It should be remembered, however, that the superficial part of the capsule is the toughest part, and it is inadvisable to push too hard with a blunt needle, as this may **cause a rupture** in the depths where the wall is thinnest. Sometimes the decompression may bulge so much that the scar becomes very thin, despite frequent lumbar punctures and dehydration; the pyograms will show considerable enlargement of the abscess, and in these cases aspiration with a sharp needle should be repeated as often as is necessary to maintain the integrity of the wound.

7. Extirpation. When it has been decided that the abscess is ready for excision, the technical problem is that of the removal of a subcortical cystic tumour of the brain, complicated by the fact that the contents of the cyst are usually infected and that the wall is not of uniform thickness and durability. The details of technique are probably not of wide interest, and a brief description should be sufficient.

The choice of anaesthetic is of some importance. By the time these cases come to operation there is usually some increase in intracranial pressure, and our preference has been for local anaesthesia, as any inhalation anaesthetic, such as gas, oxygen and ether, tends to raise

the pressure, increase venous congestion, and generally render the operation more difficult. The local infiltration of novocain can be made in such a way that the operation is painless, but the patient may be restless from confinement in one position, or may object to the noise of cutting and sawing bone, and in these cases one of the intravenous anaesthetic agents, such as pentothal, may be of great help. Given properly, it seems to be a safe anaesthetic agent, and it has no effect on the intracranial pressure: if an efficient airway is maintained, the brain behaves much as under a local anaesthetic.

An osteoplastic flap is reflected, as for the exposure of a brain tumour. In the case of a frontal lobe abscess, an ordinary transfrontal flap suffices. For the temporal lobe abscesses, the flap needs to be designed so that access is adequate for the floor of the middle fossa, as the abscess is attached there (Fig. 26).

Once the flap is reflected, the dura may be so tight that some relief of tension is necessary before it can be safely opened. This may be obtained, in most cases, by tapping the opposite ventricle through a frontal or parietal burr hole. If that is not adequate, or if the ventricle cannot be found, the intravenous injection of 50 c.c. of 15 per cent. NaCl may produce the desired effect. If neither of these methods is sufficient, there

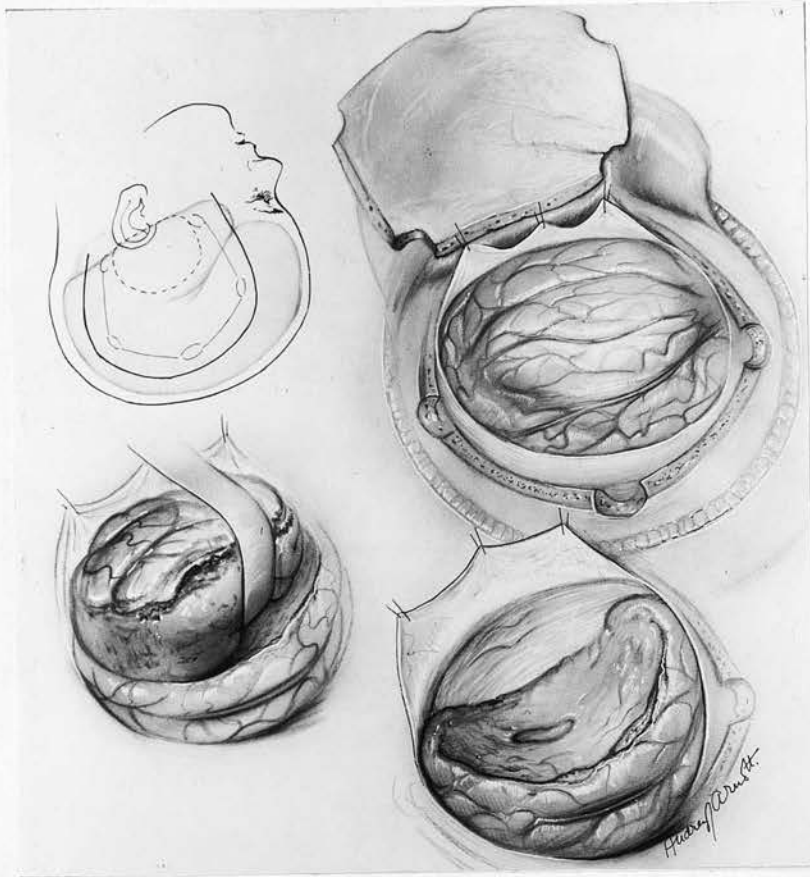


Fig. 26. Operation sketch of the removal of a left temporal abscess. The upper left figure shows the outline of the scalp and bone flap in relation to the abscess. The upper right illustrates the broadening and pallor of the convolutions over the abscess, and the upward displacement of the Sylvian vessels. The area of cortical excision and the dissection from the brain are seen in the lower left. In the lower right, the dura of the middle fossa, the site of attachment to the tegmen tympani, and the rent in the temporal horn of the ventricle are seen.

is probably a large collection of pus in the abscess, and it should be aspirated. A sharp lumbar puncture needle should be used and the pus aspirated slowly with a syringe. The removal of 10-15 c.c. may be enough to slacken the dura enough for easy opening.

The dura should be opened only over that part of the cortex which covers the abscess. It is usually possible to recognise the convolutions over the abscess, as they are broader and paler and somewhat flattened. Sometimes they may have a rather translucent greenish-yellow appearance when the capsule of the abscess is very close to the surface of the brain. If these surface manifestations are not apparent, and if the location of the abscess is not known with certainty from previous pyograms and ventriculograms, its nearest point to the surface can be determined by putting in a blunt brain needle and "feeling" for it.

Having located the wall of the abscess, it is usually advisable to excise a circle or ellipse of cortex over it. In some cases of small abscesses, it is possible to dissect through a linear incision in the cortex, and this procedure is helpful in the cases of abscesses underlying eloquent parts of the brain such as the motor cortex, left supramarginal gyrus, etc. Otherwise, the additional room afforded by the excision of cortex is very well worth

while, as it means less retraction on the surrounding brain and greater freedom of manipulation, with correspondingly less risk of rupturing the abscess.

Once the most superficial surface of the abscess is exposed, the **procedure** then is simply one of dissection from the surrounding white matter. As the brain is usually oedematous, the dissection is not very tedious, except at the point of attachment to the dura; in the case of temporal lobe abscesses, usually over the tegmen tympani; in the frontal abscesses, at some point on the posterior aspect of the frontal sinus. This attachment can be cut across close to the dura, or separated by blunt ~~dis~~section. In some cases where the abscess is attached over a wide area of the meninges it is easier to cut the dura around the attachment and strip it from the bone. This should only be done where there is reason to believe that the bony infection has completely resolved.

In the case of very large abscesses with thin walls it is often advisable to aspirate the abscess once it is exposed. This converts a large cystic tumour into a collapsed sac, and gives much more room for the dissection of the deepest part (where the wall is thinnest) and lessens the risk of an unanticipated rupture during the dissection. The procedure usually is to expose the most superficial part of the abscess by removing the overlying

cortex. As this part of the wall will probably be the thickest and toughest, it may be possible to insert a purse-string suture in a circle of 2 cm. diameter. The suture, for preference, should not penetrate the wall into the cavity of the abscess, but it is not always possible to confine its course to the wall itself. The next step is to pack off the entire field with strips of lintine soaked in an innocuous antiseptic solution, such as proflavine. The head is then covered with a towel through which a small hole is cut, to expose the surface of the abscess and the purse-string suture. An incision is then made into the abscess inside the purse-string suture, and the sucker immediately introduced to evacuate all the pus. A small pledget of wool soaked in a stronger antiseptic (such as azochloramide in triacetin) is placed in the cavity and the purse-string tightened. The sucker is then replaced by a clean one, and the superficial drapery and antiseptic packs are removed. This procedure is probably not without risk of contamination of the field, and yet if it is carried out as outlined above the risk must be a small one, as we have not had any recurrent infections which could be assigned to it. For that matter, rupture of the abscess does not seem to be very dangerous, as on several occasions we have allowed a few drops of pus to escape from a rupture which was not

noticed for a few seconds before the sucker could be got to it. In these cases we have hastily sucked out the loculus from which the pus was escaping, packed it with cotton wool soaked in azochloramide, changed the packing around the field, re-sterilised the instruments, and finally irrigated the field with proflavine; and no harm has seemed to come of it. Nevertheless, it is a worrying technical breach which should be avoided, and it seems that controlled aspiration lessens the risk.

In some cases traction stitches carefully inserted into a thick part of the wall are a help in the dissection of the depths.

Once the abscess is excised, the brain should become "slack", as after the removal of a tumour. It is important to work in a field as free from blood as possible, and to be very thorough in the haemostasis of the cavity in the brain resulting from the removal of the abscess. Untidy operating means bruised brain and a possible nidus for any lurking infection. Similarly, if any clot is allowed to collect in the cavity, the risk of recurrent infection is enhanced. We have found hydrogen peroxide a useful haemostatic agent for troublesome oozing; a pledget of wool soaked in peroxide is left in the cavity for a few minutes, and even though this does not stop all of the bleeding points it usually reveals them

discretely enough to allow them to be picked up and stopped with the endothermy.

No attempt is made to close the dura completely. In some cases of superficial abscesses adherent to the dura a considerable area of dura may have to be sacrificed, but in the presence of a potentially infected field we have not felt that the risk of inserting a fascial graft was justifiable.

The wound is closed without drainage in the usual manner for an osteoplastic flap. The dressing is done on the following day, and if there is any large collection of fluid under the flap it is aspirated through a brain needle inserted through the suture line. Any tight stitches are cut and all of the stitches are removed on the fifth day. Daily lumbar punctures are done, on each occasion removing enough fluid to bring the pressure down to normal.

8. Chemotherapy. Drugs of the sulphonamide group are probably of greatest value in the prevention and treatment of the meningitis which may occur during the incubation or treatment of the brain abscess. The need for their use depends on the clinical state and on the examination of the cerebrospinal fluid. If they produce general toxic effects or adverse effects on the leucocyte system, they should be discontinued for the time being, if the general condition of the patient allows it. As to which

drugs should be used, the best guide is an in vitro test of the sensitivity of the responsible organism to the drug. At present, the ones most commonly employed are sulphadiazine and sulphapyridine, as these preparations seem to attain more satisfactory concentrations in the cerebrospinal fluid than any others, and are effective against a wide range of organisms. They are also tolerated well by the patient and can be taken for long periods if necessary.

Cerebellar Abscesses. The technique described above has been chiefly concerned with abscess of the cerebral hemisphere. It has been a disappointment that the same principles of treatment have not been equally successful in cerebellar abscesses, of which there were 7 in this series, with only 2 recoveries.

Of the 5 fatal cases, 2 died of meningitis after extirpation of the abscess; one died after cerebellar decompression; one died after aspiration when the patient was in extremis; and one was undiagnosed and untreated. Of the two which recovered, one was treated by decompression and repeated aspiration, and one was a fluid tuberculous abscess which was first aspirated and decompressed and ultimately extirpated.

A number of factors contribute to the hazards of an abscess below the tentorium: a relatively small abscess in the cerebellum surrounded by a little oedema will

produce gross pressure disturbances, e.g., tonsillar herniation, obstruction at the incisura tentorii and a rapid increase of intraventricular pressure. The nearness of the abscess to the brain stem renders these patients liable to the rapid onset of medullary compression or oedema which may be fatal within a few minutes. The technical difficulty of approach is another considerable factor: five of the seven cases in this group were due to mastoid disease, and it has been seen that such abscesses are usually attached to the posterior surface of the petrous bone, in the very depths of the posterior fossa. In two of these cases the abscess had attained a sufficiently thick wall to justify extirpation, and the dissection proceeded smoothly until the attachment to the petrous bone was reached, when it was found that the wall abruptly became soft and spongy, exuding beads of pus. As this could not be foreseen until the late stages of the operation it was impossible to avoid disseminating the infection, and in both cases the outcome was fatal. This is a feature of the pathology of cerebellar abscesses which we have not encountered in abscesses above the tentorium.

It seems that the following possibilities of treatment have to be considered:

- (1) Aspiration.

- (2) Cerebellar decompression, with or without opening the dura, combined with aspiration.

(3) Extirpation.

(4) Continuous drainage.

(5) "Sterilisation" of the abscess by aspiration and instillation of some bactericidal agent such as penicillin.

(1) Aspiration. It has been said that some cases of supratentorial abscess resolve simply with aspiration. There seems to be no reason why the same thing should not happen with cerebellar abscesses, and Schreiber (29) has reported a series of cases of otogenic cerebellar abscesses so treated, in some of which the cure was permanent. It is apparently impossible to predict the outcome with any degree of certainty, and whereas in the case of frontal or temporal abscesses an abscess which is resolving with aspiration can be decompressed or extirpated, the risks of such procedures in cerebellar abscesses are much greater. As it is a simple procedure which may be successful it is worth a trial.

(2) Perhaps a better chance is afforded by a suboccipital craniotomy and aspiration of the abscess through the unopened dura. How much of a decompression effect is obtained by removing the bone and not opening the dura is questionable, but certainly in some cases of brain tumour the effect is considerable, as judged by clinical and manometric observations. In some cases of cerebellar abscess one has seen the unopened dura bulge noticeably after removal of the occipital bone, and it is probable that even a slight decompression afforded by stretching of the thin dura in the

posterior fossa is of value.

One of the successful cases in this series was treated in this manner (Case 12), viz., by craniotomy, aspiration through the unopened dura, instillation of thorotrast, and a second aspiration. Together with the clinical improvement in the successful case, there was a progressive shrinking in the thorotrast-encrusted capsule which could be followed by serial X-rays.

(3) In one case neither aspiration nor aspiration / decompression were effective, and an attempt was made to extirpate the abscess. As in the cases mentioned above, the early stages of the operation were encouraging because the capsule was quite thick and it was possible to effect a clean dissection until the attachment of the abscess to the petrous bone was reached. Here there were purulent granulations which contaminated the field and led to the formation of subsequent abscesses, one of which ultimately proved fatal. X

The only successful case of extirpation of a cerebellar abscess in this series was an unusual fluid tubercular abscess, not attached to the petrous bone, and thus for technical purposes presenting a different problem, viz., that of excision of an intracerebellar cystic tumour.

At the present stage it seems that exirpation is too hazardous to be generally recommended as a method of treatment.

(4) A fourth possibility is continuous drainage, either by a suitably-placed drainage tube or by "open" drainage by the fungus method. Drainage by a tube carries the same risks and uncertainties as in abscesses above the tentorium (v.s.), but there is no doubt that it is effective in some cases. No success met this method in this series, but there are many reports of isolated cases which have had a favourable outcome. The route of introduction of the tube is probably important: Macewen had four successes out of seven treated cases, and this may be accounted for by the fact that he placed the drainage tube in the abscess through the mastoid wound, and thus had to traverse a minimum depth of cerebellar tissue. Although cerebellar abscesses are not very common, it would be interesting and valuable to treat a series of cases in this manner.

An elaboration of this method which may have its uses is in the creation of a fungus in relation to the mastoid wound by opening the dura behind the genu of the transverse sinus and allowing the cerebellum to protrude through the opening. After 2-3 days, when the subarachnoid space has been sealed off around the margins of the fungus, the fundus can be cut out with the endothermy point, the capsule of the abscess explored and opened, and slightly packed to keep it open. The protrusion of the fungus

can be controlled by lumbar puncture drainage, and there is the additional benefit of a decompression effect in this type of operation which is lacking in the case of "closed" drainage by a tube. Because of the depth and fixity of the abscess, it is unlikely that it would be extruded, but by being kept open it is possible that the cavity may ultimately become sterile and the discharge cease. When that happens, the fungus would epithelialise fairly quickly. This method might be of particular use in cases in which the mastoid wound fails to heal before some intervention is necessary to deal with the abscess.

(5) A fifth method of treatment which may prove to be of value is repeated aspiration of the abscess and instillation of some powerful bacteriostatic agent such as penicillin. In one case of frontal lobe abscess we have succeeded in "sterilising" the abscess with penicillin instillations, although it was necessary to repeat the aspirations on eight or ten occasions. Repeated aspiration is generally more difficult in cerebellar than in cerebral abscesses because the former are generally smaller and more deeply situated. The risk of spreading the infection by unsuccessful needling has already been mentioned.

Although the potentialities of penicillin have not been fully explored, it may prove to be effective in preventing a spread of infection or a recurrence after extirpation

operations (p. 147). If so, extirpation would become the method of choice, as in cerebral abscesses, and for the same reasons.

VIII. Priority of Treatment.

In the case of abscess consequent on mastoid or frontal sinus disease, the question often arises as to which condition should be treated first, or whether they should both be treated at the same time. This problem arises more frequently with otogenic abscesses than with ones due to sinus infection, because a temporal lobe or cerebellar abscess is usually accompanied by a subacute or chronic mastoid infection, whereas an acute inflammation of the frontal sinus has usually resolved completely by the time the brain abscess demands treatment.

The answer is surely that the most urgent condition should be treated first. If the mastoid infection is in an acute stage, as witnessed by pain, fever, and local signs of inflammation, it should be dealt with, even though there may be grounds for thinking that there is a brain abscess too. The indication for treating the brain abscess first is in those cases of chronic mastoid infections where the symptoms are chiefly referable to the brain abscess, i.e., headache, stupor, focal signs, etc. The danger here is that the patient may die from a rupture of the abscess while the mastoid is being dealt with,

especially if that means having an inhalation anaesthetic. In these cases, a burr hole over the abscess will permit an aspiration and this may tide the patient over until the mastoid can be dealt with.

If a mastoid operation has to be done prior to or during the treatment of a brain abscess, there seems to be a good deal to be said for the transmeatal operation: it apparently affords adequate exposure, and it leaves the scalp free of an infected wound if a craniotomy has to be done before the mastoid wound has healed. This applies to the scalp incisions used for temporal lobe and cerebellar abscesses.

In some cases the question will arise of tapping the brain abscess through the mastoid incision at the time of the mastoid operation. An exploring needle passed through the mastoid field will certainly be infected, and as it has to traverse the subarachnoid space and probably some uninfected part of the brain (either temporal lobe or cerebellum) there is considerable risk of introducing infection. We feel very strongly that this should never be done and that it is preferable to make a clean incision and a burr hole in the squamous temporal bone, as described above (Fig. 25). We have recently had two cases of fungus formation occurring through a mastoid wound in which the dura had been pierced in an unsuccessful search for

pus in the temporal lobe (Fig. 8). Such a complication adds enormously to the difficulties and dangers of treatment and could have been prevented by a clean aspiration through a separate burr hole.

IX. Test of Cure.

When should a case of brain abscess be dismissed as cured? From what has been said, it will be seen that there may be several stages in the course of treatment when the patient looks and feels well, has very little in the way of neurological abnormalities, and may be on the point of being discharged from hospital when there is a recurrence of headache and stupor demanding further treatment. There have been instances (especially of cerebellar abscesses) in which the patient has been discharged from hospital and readmitted in a moribund state some weeks later, with a reaccumulation of an abscess which has been drained (or aspirated), or meningitis from rupture of such an abscess. How are we to know that the abscess is finally cured?

In the case of an abscess treated by extirpation, the wound should have healed by first intention and the scar should be sound. There is generally no difficulty in telling about that. The cerebrospinal fluid pressure should have returned to normal, and papilloedema should

have subsided. More important still, the cerebrospinal fluid should be normal on analysis, especially with regard to protein and cell content. If there is any persistent abnormality in the cerebrospinal fluid, i.e., a slight increase in the protein content (e.g., to 75 mgm.) and even a slight pleocytosis (e.g., 6-6 cells), some infection persists and the case should not be allowed to escape observation. In some cases this may mean a recurrent abscess at the site of operation, but in others it may be a benign meningeal reaction which may take a long time to resolve. The only way to be certain is to do a ventriculogram: if the abscess has been cured and there is no recurrence, the ventricle will be slightly dilated in the vicinity of the scar. If it is a low-grade chronic meningeal reaction, both ventricles will be slightly dilated.

In the case of an abscess which has not been excised, these tests of cure should be applied very strictly. If a continuous drainage method (or aspiration) has been employed, the wound should be healed and the cerebrospinal fluid should have returned to normal. If thorotrast has been used, the abscess cavity should have shrunk to a very small size, and usually it will have assumed a crenellated outline, showing that there is no tension within the abscess (Figs. 38 and 39). In these cases a

ventriculogram should be done before the patient is finally dismissed, and he should be examined at regular intervals thereafter to be certain that he is not developing papilloedema and secondary optic atrophy without other symptoms.

X. Results.

It has been possible to follow the 24 cases which recovered for periods of from one to four years after operation. Two patients developed epilepsy (v.i.) which did not respond to sedative treatment, and both died in status epilepticus. The remaining 22 patients are well and all are engaged in some useful occupation, or, in the case of children, are proceeding normally with their education.

The completeness of the recovery from neurological abnormalities (aphasia, field defects, hemiplegia, etc.) depends on how much and what part of the brain has been destroyed by the abscess or by operative intervention. Some of these abnormalities deserve special mention.

Aphasia. In 8 of the 22 cases of cerebral abscess which recovered, the lesion was in the speech-dominant hemisphere. Five of these had varying degrees of aphasia before treatment, and in each case the recovery of the language function was complete. In 4 of these cases the abscess was extirpated; in the other it was treated by decompression and aspiration.

Visual Defects. One patient (mentioned above) was blind on admission owing to secondary optic atrophy which had developed with a chronic left frontal abscess. There was, of course, no return of vision after extirpation of the abscess, but she has been trained to operate a knitting machine and is thus usefully employed. Another patient's acuity was reduced to less than 6/60 in each eye, but he returned to work as a labourer one month after discharge from hospital and has not missed a day in the last two years.

Most of the temporal lobe abscesses have been left with an incomplete upper quadrant hemianopia, but none of them is aware of it, and it seems to occasion no inconvenience. In one case of a large temporal lobe abscess in a child, the hemianopia is complete, except for macula sparing, but it does not seem to disturb him and he is doing well at school. In another case of multiple parieto-occipital abscesses there was a residual homonymous hemianopia associated with a gross disorder of orientation which required a good deal of readjustment. This case has been described fully elsewhere, but it is worth noting that this patient has returned to his former work of a skilled technician and is doing it satisfactorily.

Hemiplegia, Sensory Defects, etc. In only one case has there been any significant motor or sensory defect after treatment. This was a child with a large multilocular abscess in the right Rolandic region after a

compound depressed fracture. She had a complete hemiplegia prior to operation, but has recovered sufficient power in the lower limb to walk unaided, and this improvement continues. The upper limb is of very little use to her, although there are feeble movements at all joints. The cortical type of sensory defect probably contributes to her disability, which is that of an incomplete hemiplegia.

"Cerebellar Signs." In the two cases of cerebellar abscess which recovered, both had very gross abnormalities prior to operation and recovery in each case was complete. It was not possible to demonstrate any residual cerebellar defect at all.

"Mental Symptoms." Of the 22 patients with cerebral abscesses, only one (Case 19) showed any evidence of intellectual impairment after operation. In this case an abscess in the right temporal lobe was consequent on an infection of the maxilla and frontal bone. It was a long, complicated illness, and a ventriculogram done as a final test of cure showed a considerable bilateral ventricular dilatation. The psychic change was perhaps not so much an intellectual as a moral one: formal intelligence tests showed that he was still a superior adult, and he returned to skilled actuarial work which he does as well as before his illness; yet his friends

and relations notice that he has changed, and for the worse: he has become somewhat selfish, conceited, unpunctual in appointments, lacking in consideration for others, and something of a bore, all traits foreign to his nature before his illness. It is possible that these changes might have passed unremarked in a person of less intellectual development, but they are none the less real or important. As the extirpation of the abscess in this case entailed only the excision of the anterior part of the right temporal lobe, notoriously a "silent area", the changes are probably a combined result of toxic neuronal effects and hydrocephalus.

It might have been thought that the frontal lobe abscesses would have produced some mental deterioration, but in 11 of the 22 cases the frontal lobe was involved and in none of them was there any evidence of deterioration.

Epilepsy remains the most disturbing sequel because of its frequency and unpredictability. Of the 33 patients with cerebral abscesses in this series, 7 had epileptic attacks of one kind or another during the incubation of the abscess. Of the 22 patients who recovered, 3 have had a recurrence of the epilepsy over a period of from one to four years, and two of them have died in status epilepticus, one 18 months and the other 3 years after operation. Both were cases of right frontal abscess

due to acute frontal sinusitis, and both were treated by extirpation. Until their final bout of status they had only occasional isolated fits, and they had regular phenobarbital treatment from the time of operation. An autopsy was done in one case, and there was only the operative scar in the right frontal lobe without any evidence of infection. In the other case the patient had been in perfect health until the final bout of status, and the cerebrospinal fluid was normal as to pressure and content just before death.

Although there are but two cases, I know of one other in which the extirpation of a right frontal abscess was followed by fatal status epilepticus. At the autopsy again ~~there was nothing~~ except a healthy scar in the right frontal lobe. This is an uncommon sequel of operations for the removal of brain tumours, but I have seen it occur after a head injury: a shell fragment embedded in the right frontal lobe during the first World War caused no symptoms until the fatal attack of status 25 years later. At the autopsy the metallic fragment was found embedded in a healthy scar, and there was no evidence of infection or of increased pressure. The frontal lobe thus appears to be especially prone to this response.

Three of the patients who had fits during the incubation period have had none since the abscess was treated.

These results are very similar to those in the London Hospital series (Table II) in which there were 33 cerebral abscesses. Six of these had epileptic manifestations prior to treatment, and in 3 cases the epilepsy persisted after treatment. Two of these cases were treated by extirpation and one by marsupialisation. In the latter and one of the former, there were frequent attacks with occasional bouts of status epilepticus which, however, responded to sedative treatment.

Epilepsy may thus mar an otherwise good result, and it is a practice in this clinic to advise that all patients who have recovered from a cerebral abscess be kept on sedative (phenobarbital) treatment for at least three or four years.

B I B L I O G R A P H Y

1. Macewen, W. (1893) Pyogenic Diseases of the Brain and Spinal Cord. Maclehose, Glasgow.
2. Gull, W. (1857) Abscess of the Brain. Guy's Hospital Reports, Third Series, vol.iii, pp.261-311 (London).
3. Sajou's Annual (quoted by Starr, infra 4).
4. Starr, M.A. (1893) Brain Surgery. Wm.Wood & Co., New York.
5. Elkington, J.St.C. (1939) Cerebral Abscess. St.Thomas's Hospital Reports, vol.iv, pp.76-97. (London).
6. Evans, W. (1931) Pathology and Etiology of Brain Abscess. Lancet i : 1231-1235; 1289-1293. (London).
7. Cameron, H.C. (1907) A Note on So-Called "Metastatic Pulmonary Abscess". Guy's Hospital Reports, vol.lxi, 69-85.
8. Ascroft, P.B. (1943) Treatment of Head Wounds due to Missiles ; Analysis of 500 cases. Lancet ii:211-218.
9. O'Connell, J.E.A. (1943) Traumatic Cerebral Fungus. British Journal of Surgery, vol. xxx, no.119, pp.201-212. (London).
10. Trevor Ropre, T.D. (1944) A Case of Cavernous Sinus Thrombosis. British Medical Journal, i:255. (London).
11. Falconer, M.A., McFarlan, A.M., and Russell, D.S. (1943) Experimental Brain Abscesses in the Rabbit. British Journal of Surgery vol.xxx, No.119, p.245.
12. King, J.E.J. (1936) Brain Abscess. Annals of Surgery, 103:647-668. (Philadelphia, U.S.A.)
13. Lebert, G. (1856) Hirnabscesse. Archiv. fur pathologische anat. bd.x.
14. Grant, F.C. (1932) Mortality from Abscess of the Brain. Journal of the American Medical Association, 99:550-556.
15. McFarlan, A.M. (1943) Bacteriology of Brain Abscess. British Medical Journal, 2:643.
16. Alpers, B.J. (1939) Abscess of Brain : Relation of Histologic to Clinical Features. Archives of Otolaryngology, 29:119-222.
17. Grant, F.C. (1941) Brain Abscess : Collective Review. International Abstracts of Surgery, 72:118-138.
18. Kahn, E.A. (1939) Treatment of Encapsulated Abscess of Brain : visualisation by Colloidal Thorium Dioxide. Archives of Neurology and Psychiatry, 41:158-165.
19. Brain, W.R. (1941) Visual Disorientation with Special Reference to Lesions of the Right Cerebral Hemisphere. Brain, 64:244-272.
20. Dudgeon, L.S. and Patrick, C.V. (1927) A New Method for the Rapid Microscopical Diagnosis of Tumours. British Journal of Surgery, xv, 250.

21. Russell, D.S., Krayenbuhl, H., and Cairns, H. (1936) Wet Film Technique in Histological Diagnosis of Intracranial Tumours : Rapid Method. *Journal of Pathology and Bacteriology*, 45:501-505.
22. Russell, D.S. and Beck, D.J.K. (1944) Discussion on Venous Thrombosis in the Central Nervous System. *Proceedings of the Royal Society of Medicine*, vol.37, p.388.
23. Symonds, C.P. (1931) Otitic Hydrocephalus. *Brain* 54:55-71.
24. Pennybacker, J. (1944) Intracranial Venous Obstruction. *Transactions of the Ophthalmological Society* (in press).
25. Martin, J.P. and Sheehan, H.L. (1941) Primary Thrombosis of Cerebral Veins (Following Childbirth). *British Medical Journal* 1:349-353.
26. Horrax, G. (1934) A Method for the Treatment of Certain Encapsulated Brain Abscesses. *Surgical Clinics of North America*, vol.14, 1179-1186.
27. King, J.E.J. (1924) The Treatment of Brain Abscess by Unroofing and Temporary Herniation of Abscess Cavity with the Avoidance of Usual Drainage Methods. *Surgery, Gynaecology, and Obstetrics*, vol.39, 554-568.
28. Vincent, C., David, M. and Askanasy, H. (1937) Sur une Methode de Traitement des Absces subaigus et Chroniques des Hemispheres Cerebraux. *Journal de Chirurgie*, 49:1-46.
29. Schreiber, F. (1941) Cerebellar Abscesses of Otitic Origin in 9 children (8 recoveries). (*American Annals of Surgery*, 114:330-335).

CASE REPORTS

INDEX of CASE REPORTS.

.....

Abscesses due to Pulmonary Infections :

Case 1	page	160
Case 2		165
Case 3		168
Case 4		170

Abscess due to Infective Endocarditis

Case 5		174
--------	-------	--	-----

Temporal Lobe Abscesses (Otogenic)

Case 8		189
Case 9		194
Case 10		198
Case 11		201

Cerebellar Abscesses (Otogenic)

Case 12		204
Case 13		210

Abscess due to Frontal Sinusitis

Case 14		215
---------	-------	--	-----

Frontal Abscess due to Penetrating Wound

Case 15		219
---------	-------	--	-----

Frontal Abscess due to Compound Fracture of Skull

Case 16		223
---------	-------	--	-----

Abscesses due to Infections of Face, Scalp and Skull

Case 17		227
Case 18		230
Case 19		234
Case 20	(Cavernous Sinus Thrombosis) ..		241

Case 1 . Right frontal abscess due to chronic empyema thoracis. Aspiration of abscess, followed by decompression, opening of dura, and eventual removal of large multilocular abscess. Subsequent development of left temporal abscess, treated by aspiration, decompression and eventual removal. Death from untreated left frontal abscess.

Mr. W.R., aet. 28 (R.I.19741) admitted 28th. November, 1941. He had been in good health until June, 1941, when he developed a left-sided pneumonia followed by empyema. This was treated by repeated aspiration, tube drainage in the eighth intercostal space, and eventually a modified thoracoplasty. Twelve days before admission, another drainage opening was made in the eleventh intercostal space. Five days later (22nd. November, 1941) he had a generalised epileptic attack preceded by a momentary feeling of stiffness of the left arm. He recovered from this but on 26th. November he had an attack characterised by clonic movements of the left arm and left side of the face, but there was no loss of consciousness. Later in the same day he had a similar attack which was followed by complete paralysis of the left arm and left side of the face. After this attack and for the first time he complained of slight generalised headache.

On admission, he was very wasted but there was no rise of temperature or pulse rate. There were two sinuses in the left posterior thoracic wall draining thick yellow pus. The respiratory rate was 26/min. He was conscious and rational although slightly drowsy. There was no papilloedema and the visual fields were full. Conjugate movement of the eyes to the left was defective. There was marked weakness of the left side of the face, complete paralysis of the left upper limb, and a moderate weakness of the left lower limb.

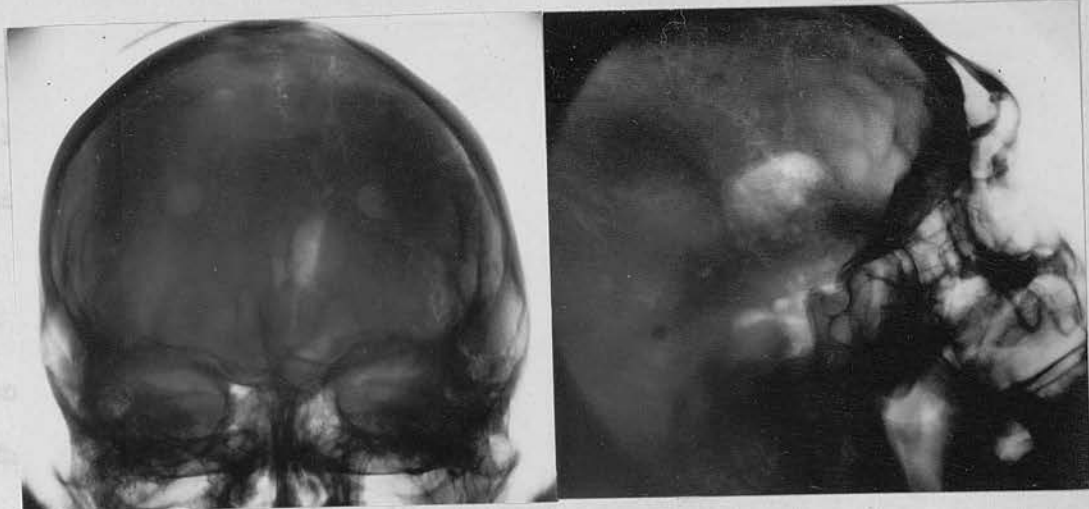
The tendon reflexes were slightly increased in the left limbs, the left abdominal reflexes were absent, but both plantar responses were flexor.

1st. December, 1941 - lumbar puncture pressure 120 m.m. The cerebro-spinal fluid was clear and colourless containing 50 mgm. protein and 2 cells. A ventriculogram done on this day showed that both ventricles were filled; there was no local deformity but the whole ventricular system was displaced to the left side, (Fig. 27). Because of the absence of local deformity it was suggested that he was suffering from infective thrombophlebitis of the right hemisphere, the displacement being due to oedema of the hemisphere.

During the next two weeks he became a little more drowsy and the left hemiplegia became complete. He also developed partial oculo-motor paralysis on the right side.

16th. December, 1941 - he was much more drowsy, almost comatose, and was incontinent of urine and faeces for the first time. The spinal fluid pressure was 250 m.m., the fluid contained 60 mgm. protein and 2 cells. A right frontal burr hole was made and a small abscess was found in the posterior part of the frontal lobe from which 5 c.c. of thick offensive-smelling pus were aspirated. One c.c. of thorotrast was put into the cavity (Fig. 27). From the pus and anaerobic streptococcus and a fusiform bacillus were cultured. He was much more alert immediately after the aspiration, but by 18th. December he had relapsed into a deep stupor from which he could barely be roused by painful stimuli. He was again incontinent and swallowing was impossible.

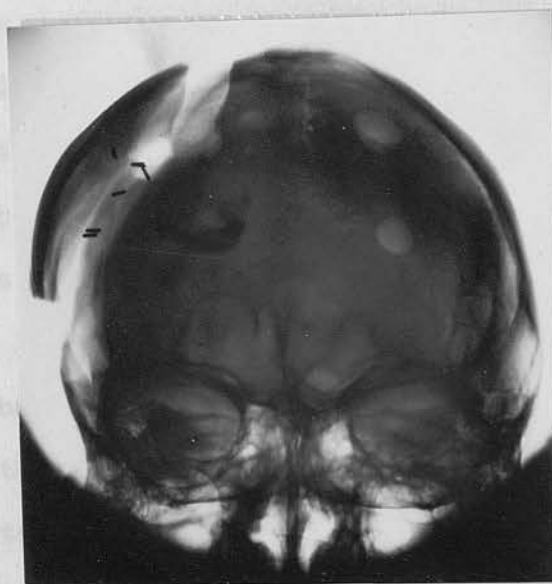
FIGURE 27 overleaf



Case 1. Ventriculogram : note displacement to left side, but absence of deformity.



Pyrogram after initial aspiration and instillation of thorotrast on 16th. December, 1941.



Pyrogram one week after decompression. Note riding of bone flap, and displacement of abscess into decompression



Multilocular Abscess removed on 8th. January, 1942.

19th. December - a right lateral osteoplastic flap was reflected under local anesthesia. The dura was not tight. When it was opened, the leptomeninges looked healthy but the abscess could be seen presenting on the surface of the brain in the Rolandic area. The dura was left widely open and the cortex was covered by thin strips of gutta percha. The bone flap was replaced and the scalp wound closed.

There was considerable improvement following the decompression. He was more alert, less often incontinent, swallowing well, and speaking rationally. Lumbar punctures were done every day, the pressure varying between 140 m.m. and 230 m.m. The removal of 10-20 c.c. of spinal fluid would slacken the tension on the flap and the wound healed normally. X-rays showed that the flap was riding, the thorotrast shadow was increasing in size and tending to be displaced into the decompression (Fig. 27). There was no change in the neurological picture except that the oculo-motor palsy cleared up.

Two weeks after the decompression he again began to get very drowsy and the bone flap and decompression were bulging to such an extent that the scar was beginning to get thin in places.

By 8th. January, 1942, it was felt that the abscess was at least 47 days old and that it might be possible to remove it. The flap was re-elevated, and although the dura was adherent to the cortex over the abscess, the cortex protected by the gutta percha was not adherent to the bone flap, nor had it been at all bruised by being herniated through the dural opening. A large bilocular abscess was dissected out intact (fig. 27), and although the surrounding brain was oedematous it became quite slack after the abscess was removed.

There was again a great improvement in his general condition and state of alertness. The wound healed normally and five days after the operation he asked to be allowed up. This improvement continued until twelve days after operation.

On the 20th. January, it was found that he had suddenly become aphasic and developed a profound weakness of the right side of the face and right upper limb. There was no rise of temperature nor any signs of meningitis. The spinal fluid pressure was normal, the fluid clear and colourless containing 80 mgm. protein and 10 lymphocytes. During the next two weeks he became progressively more drowsy and aphasic, and the right hemiplegia was almost complete.

On 3rd. February, he was comatose and the right-sided decompression was bulging. A ventriculogram was done (Fig. 28) and this showed a dilated right ventricle (indicating that there was no longer any abscess formation in the right hemisphere) displaced to the right, and a filling defect in the left temporal horn. Accordingly, a left temporal burr hole was made and 8 c.c. pus were aspirated from the left temporal lobe. There was little improvement so on 5th. February a left temporal osteoplastic flap was reflected and the abscess could be seen occupying the posterior part of the temporal lobe. As at the decompression on the right side, the dura was left open and the cortex protected with strips of gutta serena tissue.

The wound healed normally and his general condition improved. On 21st. February he began to get drowsy again, the aphasia was more pronounced, and on this day he had a Jacksonian attack affecting the right side of the face, the right arm and leg. From that time there

FIGURE 28 overleaf

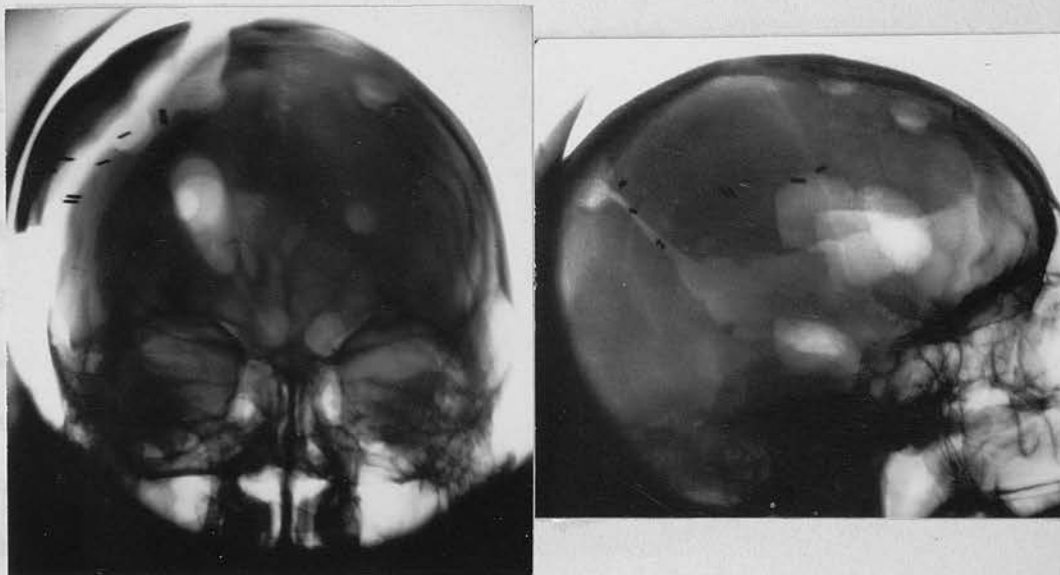


Fig. 28. Case 1 - Ventriculogram on 3rd. February showing dilatation of right ventricle and displacement of whole ventricular system to the right side. Note also the elevation of the bone flap.

The plate at the upper right shows the healthy scar in the right hemisphere at the site of the removal of the abscess seen in Fig. 27. The ventricle is drawn up into the scar, as seen in the ventriculogram above.

The untreated left frontal abscess is seen in the lower right plate.



were frequent epileptic attacks in which it appeared that he was dying.

On 9th. March, the left temporal flap was re-elevated and a large multilocular abscess weighing 117 grams was removed from the left temporal lobe . This abscess was of about the same size as that which had been removed from the right hemisphere. It was reckoned to be 48 days old. The wound healed normally, and there was some improvement in his general condition, but he remained completely aphasic and with a bilateral hemiplegia. On 23rd. March, there was some inflammation of the anterior limb of the incision and at this time he developed a fulminating respiratory infection from which he died on 26th. March.

At the autopsy, the right hemisphere was found to be free of infection. The ventricle was dilated (Fig.28) and it was clear that the right hemisphere abscess had been dealt with satisfactorily. There was some subgaleal infection of the left temporal flap, but more important was the presence of a large abscess in the left frontal lobe (Fig.28) which had not been detected. It probably began on 21st. February when he had the first right-sided Jacksonian attack.

Comment : This case shows that there is nothing inherently fatal about bronchogenic abscesses (cf. Case 4) as the one in the right hemisphere was effectively dealt with by extirpation. The continuing chest infection caused another abscess in the left temporal lobe two weeks after the removal of the one in the right hemisphere. Even though he had recovered from this one, there would have been the third metastasis in the left frontal lobe. If it had been possible to arrest the chest infection before these left hemisphere metastases , the outcome might have been different.

Case 2 . Left Fronto-Parietal Abscess (anaerobic streptococcus) due to Mild Chronic Bronchiectasis. Aspiration of Abscess. Death.

A.W., male, aet. 42 (R.I. 17167) admitted on 7th. April, 1941. He had been in good health until 5 years before when he had a gastro-enterostomy for chronic peptic ulcer and at the same operation a cholecystectomy was done. Convalescence was complicated by a pulmonary embolus in the left lung, followed by a left lobar pneumonia and empyema which resolved with repeated aspirations. In the following year, he was said to have had an abscess of the right lung which discharged into a bronchus and resolved satisfactorily.

Two years before admission an abscess developed in the left lung. This discharged into the pleural cavity and the resulting empyema was drained through a rib resection. The sinus healed in a few weeks but he was left with a persistent cough, copious purulent sputum and an occasional small hemoptysis. Chronic bronchiectasis was diagnosed and he was put on a postural drainage regime.

Despite all of this he had been in good general health until 8 days before admission when he had a fit in his office. It was not witnessed but when he was seen by his doctor an hour later, he complained of a "tight feeling" in his right hand and foot, and his speech was slightly disturbed. These symptoms cleared up within a few hours and he remained well for the next three days.

On 2d. August, 1941, he woke his wife at 6 a.m. in a fit. She said that the right side of the face and the right arm and leg were convulsed. The attack lasted for about ten minutes and when he was seen by his doctor half an hour later, there was a definite expressive aphasia but no demonstrable weakness of the face or limbs.

Later in the day he began to complain of severe headache , the temperature rose to 101 degrees, and by the evening there was definite weakness of the right arm and the right side of the face.

On the day before admission he became drowsier , the weakness of the right side was more pronounced and extended to involve the right lower limb, and the aphasia was much more pronounced.

On admission, he was conscious but very drowsy. Temperature 100 degrees, pulse rate 72/min., respirations 28/min., blood pressure 140/90. There was no neck stiffness or Kernigism. He was grossly aphasic, the only word which he could say being a meaningless "Yes". The optic fundi were normal but there appeared to be a right homonymous hemianopia. There was a complete right hemiplegia, the limbs were flaccid, the tendon reflexes equally brisk on the two sides and both plantar responses were extensor. He did not resent painful applied to the right side of the body whereas he did object on the left side by grimace and withdrawal. The spinal fluid pressure was 310 m.m., the fluid was clear and colourless containing 120 mgm. protein and 12 cells, 10 of which were neutrophils.

The diagnosis of left hemisphere abscess was in no doubt, but as there was such widespread disturbance of function (hemianopia, aphasia, hemiplegia and sensory loss) the localisation was uncertain. In such a case ventriculography rarely succeeds in demonstrating the ventricle on the affected side, so exploratory aspirations were done. From a left frontal site no pus was obtained, but another burr hole in the left parietal region disclosed a loculus containing 12 c.c. of offensive-smelling pus extending from the depths of the parietal

lobe extending to within 2 cm. of the surface of the brain. This aspiration did not improve his condition, he remained comatose, the respiratory rate went up to 60/min., and there were signs of consolidation of the left lung. He died 36 hours after the operation.

At the autopsy there was a multilocular abscess in the left fronto-parietal region (Fig. 29) . It was situated in the subcortical white matter, the cortex and leptomeninges in the sulci acting as septa which tended to divide the abscess into compartments. There was no macroscopic evidence of capsule formation , and the whole inflammatory area was poorly demarcated from the surrounding white matter which was very oedematous. There was some localised inflammation of the leptomeninges over the surface of the abscess but no diffuse meningitis. The lungs showed the changes of purulent bronchopneumonia superimposed on chronic bronchiectasis.

Comment : This was an acute abscess, the interval between the onset of cerebral symptoms and death being only ten days. The appearance of the abscess at the autopsy is characteristic of the acute stage and demonstrates the tendency for such metastatic abscesses to occur at the junction of grey and white matter. (See also Fig. 2,3 and 4).



Fig. 29. Case 2 - Multilocular left hemisphere abscess.

Note its situation at the junction of grey and white matter and the formation of septa by the sulci.

Case 3 . Multiple Cerebral Abscesses due to Chronic Bronchiectasis.
Aspiration of One Loculus. Death.

C.R., male, aet.26 (R.I. 12082) , was admitted on 27th. November, 1940. Three years before, he had had an acute left lobar pneumonia which was complicated by empyema. This was drained through a rib resection and at the end of six months the sinus had healed and he seemed to have recovered his health. Ever since this illness , he had had a persistent cough with copious purulent sputum, and the physical signs together with the X-ray appearances were characteristic of chronic bronchiectasis. Nevertheless he was able to carry on with his work as a lamp inspector at a colliery.

One month before admission, he had what was thought to be influenza : a slight rise of temperature, aching in all his limbs, and some worsening of the cough. After a few days in bed he improved to the point of getting up and about again but he did not return to work.

Ten days before admission he began to complain of a slight generalised headache. Two days later while taking refuge in an air raid shelter, he had a major epileptic attack : without warning he suddenly lost consciousness, became cyanosed and was generally convulsed for about ten minutes. He had a similar attack about an hour later, and when he recovered consciousness he complained of severe headache. It was noticed that he was unnaturally drowsy from that time. A third epileptic attack led to his admission to the Nuneaton General Hospital. In the week before his transfer to the Radcliffe Infirmary he became progressively more drowsy and when he was roused he complained of severe generalised headache. Except for one or two rises to (99) ninety nine degrees

FIGURE / 30 overleaf.

FIG. 30. Case 4 - *Salmonella* infection, showing the effect of the drug. The plate to the right was taken two weeks later, showing that the infection has been eliminated. It is not possible to distinguish between the two plates. The material is the same as in Fig. 30, following.

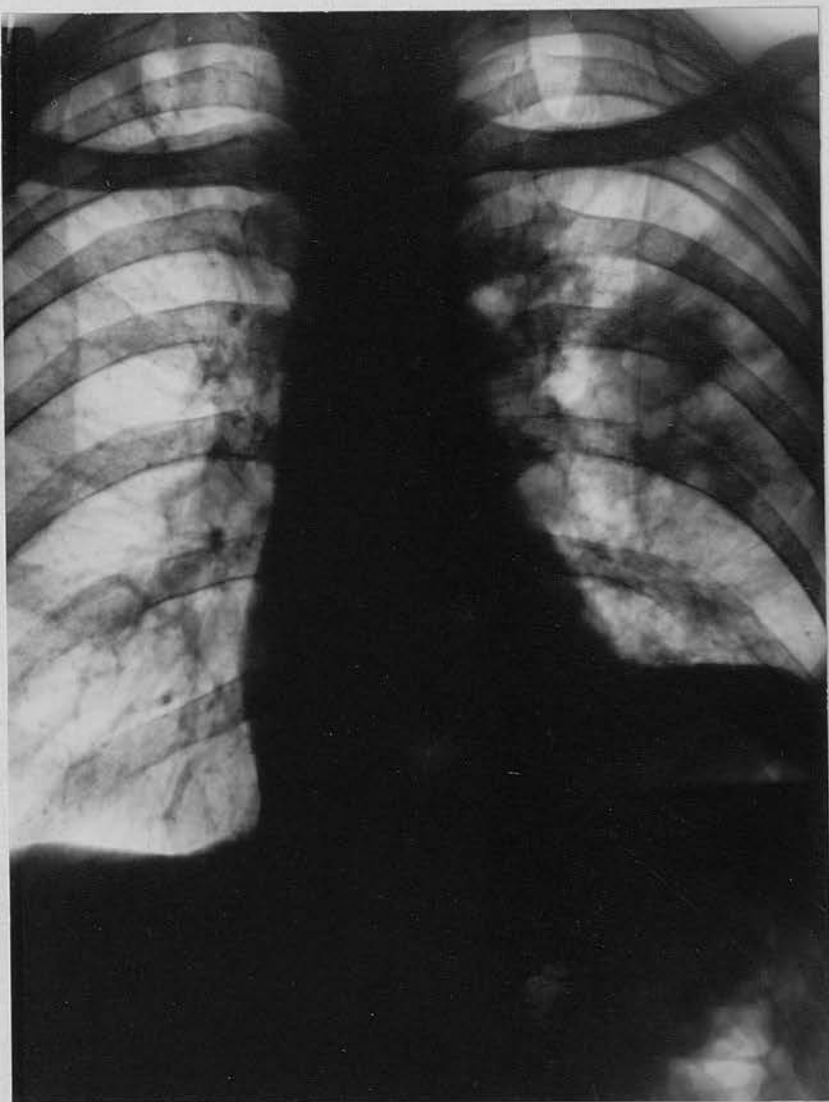


Fig. 30. Case 4 - skigram 25:ii:43, showing abscess of left lung. The plate to the right was taken two weeks later, showing that the abscess is much less distinct : it had probably discharged into a bronchus and its contents coughed up. See Fig. 30 a, following.

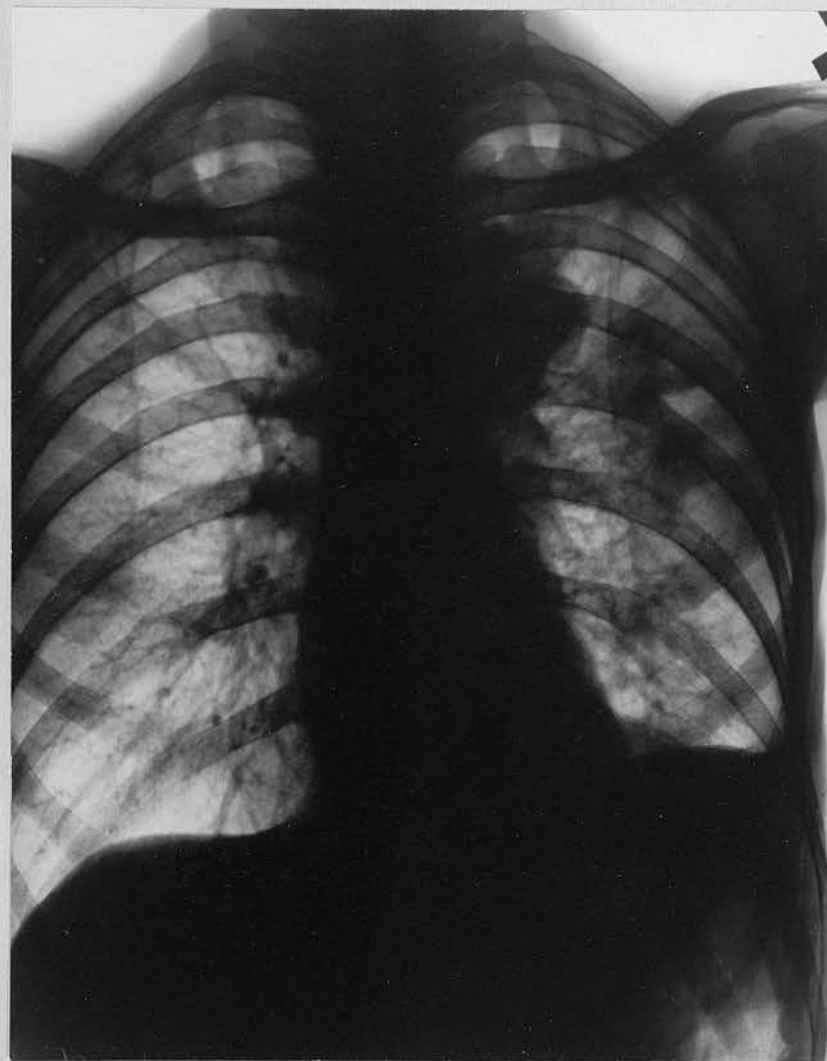


FIGURE 30 a overleaf.

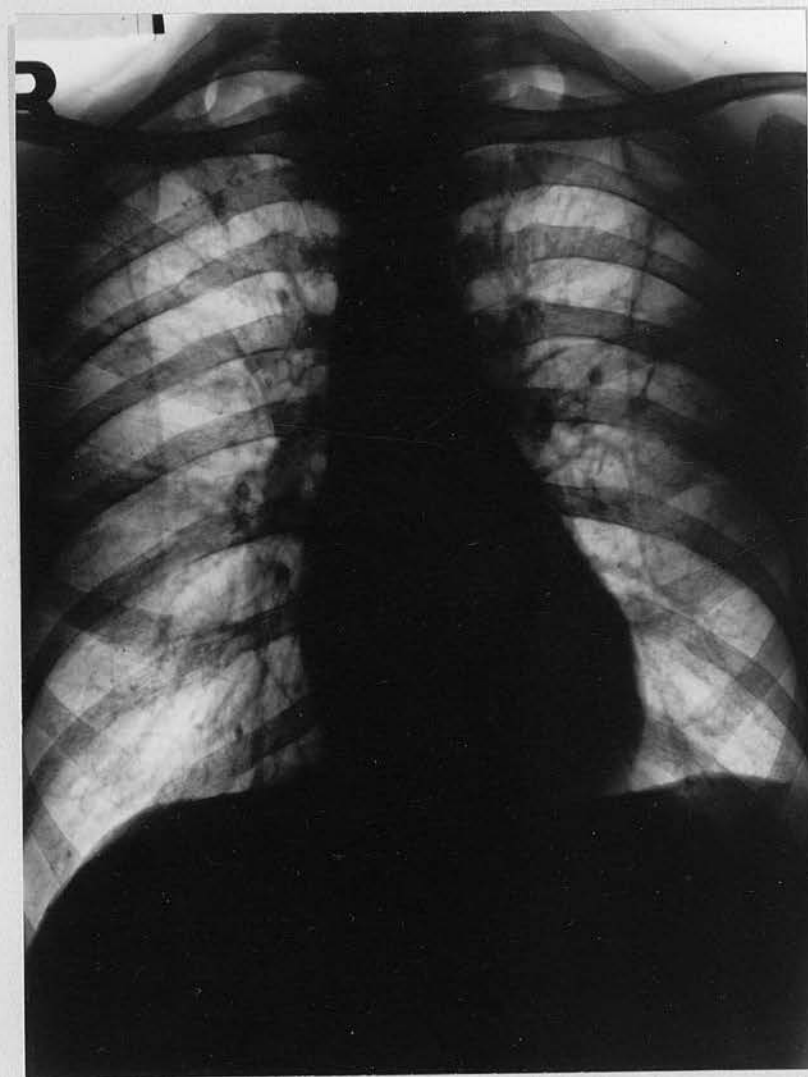
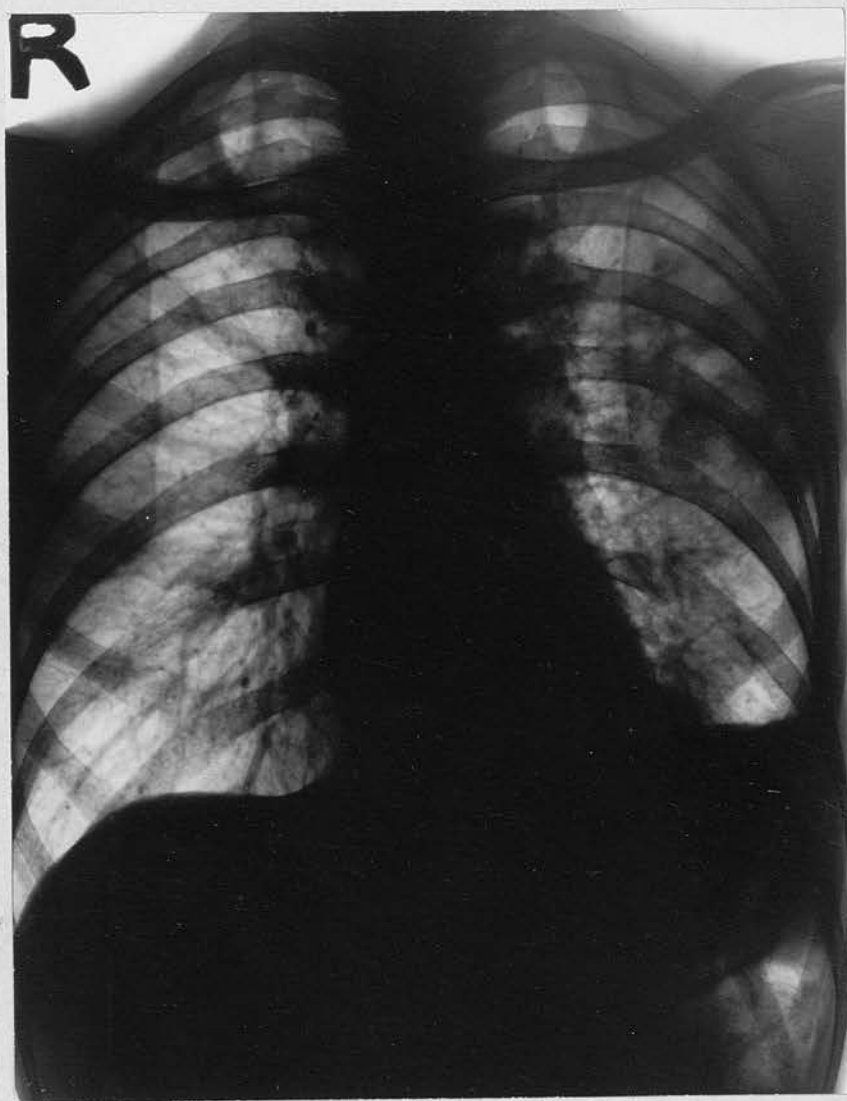


Fig. 30 a. Case 4 - showing later stages in the resolution of the left pulmonary abscess. The plate above was taken two weeks after the second in Fig. 30 ; and that to the right was taken nine months later, showing complete resolution.

Shortly afterwards, he had another fit very much like the first one again followed by a transient aphasia and weakness of the right arm and leg. He had several such fits until September, 1943, when after one of them he was left with some persisting weakness of the right arm. By the first week of October, 1943, the weakness of the arm was so marked that he had to give up his work, and it was noticed that he was dragging his right leg when he walked. At this time, he began to suffer from frontal headache. The worsening headaches and the progressive right hemiplegia led to his readmission to the City General Hospital, Leicester, on 16th. December, 1943. While under observation in this hospital he had several fits characterised by jerking of the right arm and leg without loss of consciousness, lasting for about ten minutes. The right hemiplegia became more marked and the headache more frequent and severe.

On admission to the Radcliffe Infirmary (12:i:44) he was alert and rational, complaining of a dull frontal headache. A very slight nominal aphasia could be demonstrated in formal tests, but it was not apparent in conversation. There was some swelling of the left optic disc, but the right was normal. There was no defect in the visual fields. The pupils and ocular movements were normal. There was very slight right facial weakness, but the right upper limb was almost completely paralysed, there being only feeble voluntary movements of the wrist and fingers. The right lower limb was likewise very weak, especially in the distal segments, and he could not stand or walk. The paralysed limbs were flaccid, but the tendon reflexes were exaggerated. All the abdominal reflexes were absent, the right plantar response was extensor, the left flexor. The sensory examination revealed no loss of cutaneous

sensibility, but there was loss of postural and discriminative sensibility in the right upper and lower limbs. The X-rays of the skull were normal. The cerebrospinal fluid contained 50 mgm. protein and 20 cells, mostly lymphocytes. The electro-encephalogram revealed a focus of delta activity in the left fronto-parietal region .

It was clear that there was an expanding lesion in the left Rolandic area, and the history and cerebrospinal fluid findings made a chronic abscess the most likely possibility. A ventriculogram (Fig.31) confirmed the localisation and a left lateral osteoplastic flap was reflected on 25th. January, 1944. When the dura was opened, the convolutions in the upper part of the Rolandic area were seen to be flattened and pale, and an exploring cannula encountered resistance at a depth of 1 cm. from the surface. To facilitate the dissection it was necessary to remove a circle of cortex 2.5 cm. in diameter from the posterior part of the frontal lobe over the abscess. It was then possible to dissect the abscess from the surrounding white matter and to remove it in toto. The brain became quite slack after the removal of the abscess. On section the abscess was bilocular, the walls being up to 0.4 cm. thick (Fig.31). The pus was creamy yellow ; no organisms were seen in the films, and it was sterile on culture, but histological preparations demonstrated a profuse branching mycelium which was identified as *Aspergillus*.

The operation was done under local anesthesia. He talked rationally until the time the cortical incision was made when his speech became grossly disordered. At the end of the operation, he had a very complete expressive aphasia : although quite conscious and co-operative, able to understand what was said to him, he could not form any words at all except "Yo" which seemed to be a compromise between "Yes" and "No".

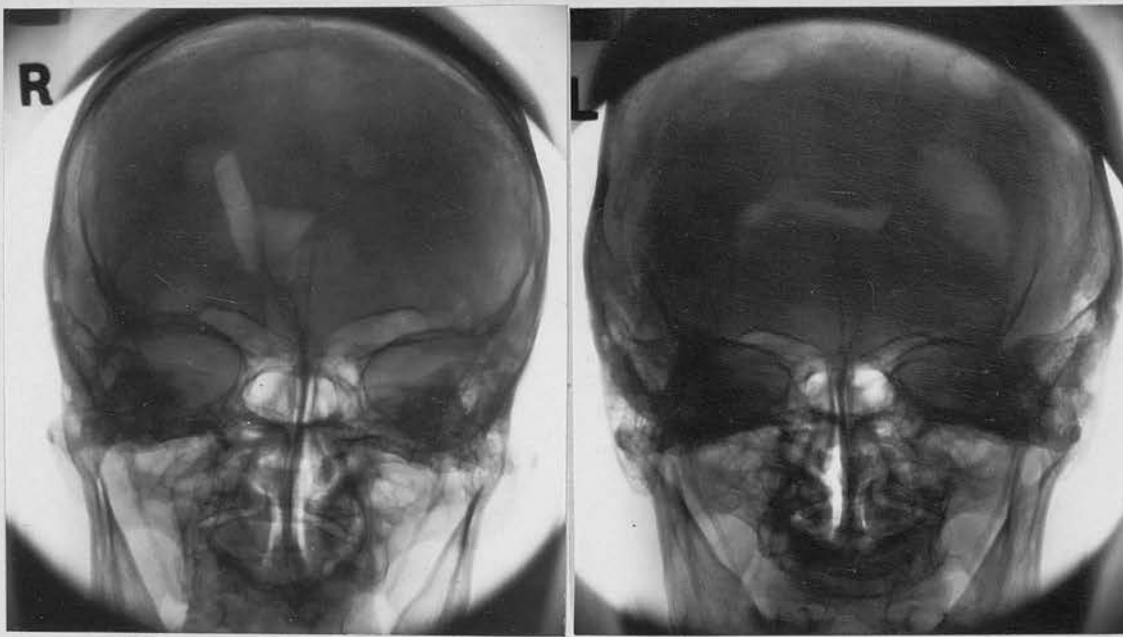
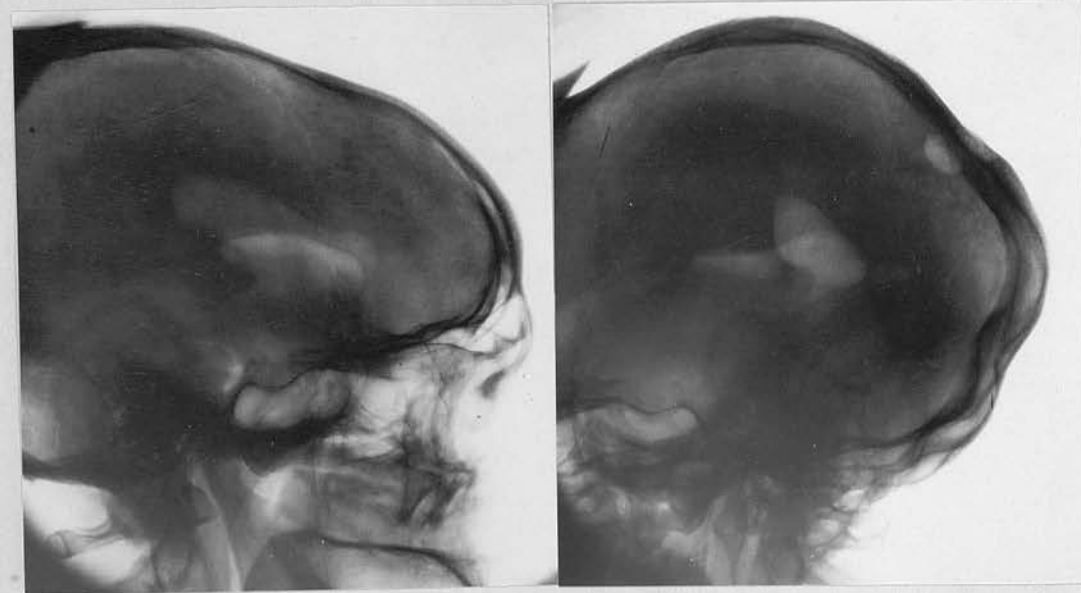


Fig. 31. Case 4 - ventriculogram on 20:i:44. Note displacement of ventricular system to right side and deformity of central part of body of left ventricle.



The abscess removed at operation on 25th. January is seen in the photograph at the right. It measured 3x4x5.5 cm. and can be seen to be covered by a thin layer of cortex.



He also had a complete right hemiplegia affecting face, arm and leg.

The wound healed normally. By 31st. January, the spinal fluid had reverted to normal (35 mgm. protein, 2 cells); the aphasia cleared up rapidly and when he was discharged on 7th. February, it was barely detectable. The facial weakness likewise had cleared up and there was some improvement in the power of the right lower limb. There was no improvement in the upper limb, and the sensory findings were unaltered. This limb is unlikely to be of any use to him, but the lower limb should recover to the point of letting him get about with only a slight limp. There had been no epileptic attacks after operation, but too short a time has elapsed to assess the result of operation on this symptom.

Comment : This is the only bronchogenic abscess in the series which has recovered. Apart from this the points of interest are (1) that the onset of cerebral symptoms occurred two and a half months after the lung abscess had resolved, although the persistent cough may have betokened some residual inflammation ; (2) that the lung abscess occurred after an abdominal operation under local anesthesia ; (3) that the abscess was a single one and not multiple as is common with metastases from the lung ; (4) the unusual nature of the organism. Fungus infections are rare in cerebral abscesses, but we have encountered actinomyces and some other unidentified fungi as part of a mixed infection in two or three cases of metastatic abscesses.

Case 5. Right Parietal Abscess (Streptococcus Viridans) due to Infective Endocarditis. Aspiration of Abscess and Osteoplastic Decompression. Death.

M.M., female aet. 20 (R.I. 21661) was admitted to the Radcliffe Infirmary on 11th. March, 1942. She had had two attacks of rheumatic fever in childhood, but seemed to have recovered satisfactorily from each of them and she led a normal healthy life until the end of October, 1941, when she had a febrile illness characterised by painful swelling of several joints. This was regarded as a recurrence of the rheumatic infection and she was kept in bed for six weeks. At the end of this time, she resumed her work but she was never really well again.

Three months before admission, she suddenly became confused at the work in a telephone exchange . She complained of not being able to see properly but more striking to observers was her difficulty in finding her way about in quite familiar surroundings. She had to be taken home and when she got there she "behaved as though she was in an entirely strange house ; she could not find her way to her room, nor did she seem to be able to find her way about among her own belongings" , although it was obvious that she was not blind . She complained of some generalised headache. She was put to bed, and for the next three or four days she vomited repeatedly. It was found that her temperature went up to 102 degrees every evening, although it was usually normal in the mornings.

Five weeks before admission, it was noticed that the left arm and leg had suddenly become paralysed and this persisted. Two weeks later, she got very drowsy and for two or three days she was unresponsive

and it was thought that she was dying. She then began to recover to her previous resting level. On one or two occasions there had been transient bouts of aphasia lasting several minutes at a time. She was right-handed.

On admission (11:iii:42) she was a well-developed young woman, but she looked very ill. She was pale with a slight cyanotic tinge on her cheeks and lips. The respiratory rate was increased to 30/min., the pulse rate was 120/min., and the daily variation in temperature was between 97 and 103 degrees. The heart was dilated and there was a loud mitral systolic murmur with a double murmur at the aortic area. B.P. 115 systolic, 50 diastolic. There were no abnormalities on clinical examination of the lungs, and the skiagrams showed only the cardiac dilatation. A blood count showed $2\frac{1}{2}$ million red cells, and 44% hemoglobin, with a leucocytosis of 16,000. Blood culture on 16:iii:42 yielded a profuse growth of streptococcus viridans. There were some erythrocytes but no organisms in catheter specimens of urine.

The neurological examination revealed bilateral papilloedema of two diopters, a complete left homonymous hemianopia, and a moderate left hemiplegia with a corresponding alteration in the reflexes. The hemiplegia was maximal in the upper limb, next most marked in the lower limb, and least in the left side of the face. There was gross impairment of all sensory modalities down the left side of the body. X-rays of the skull showed some displacement of the pineal gland to the left side, but were otherwise normal. The cerebro-spinal fluid pressure was over 300 m.m. and the fluid contained 75 mgm. protein, 124 cells (68% neutrophils). The fluid was sterile on culture.

This patient was suffering from infective endocarditis, and the cerebral lesion was most probably an embolic abscess in the posterior half of the right hemisphere. A possibility of multiple abscesses was envisaged because of the story of episodes of aphasia, one of which was witnessed while she was under observation. A ventriculogram on 13:iii:42 (Fig.32) showed no evidence of a lesion in the left hemisphere but a large abscess was encountered in the right parietal region. From this 35 c.c. of pus (from which streptococcus viridans was grown) were aspirated and 2 c.c. of thorotrast instilled. Although the endocarditis rendered the outlook very grave, it was felt that something might be learned about the technical problems of dealing with cerebral abscesses by reflecting a bone flap, opening the dura over the abscess to afford a decompression with a view to ultimate excision of the abscess. This was done on 13th. March. When the dura was opened the convolutions over the abscess were tight and had a brownish hue. The leptomeninges were of healthy appearance. The dura was left open and the exposed cortex was protected by strips of gutta serena tissue to prevent adhesions to the under aspect of the flap.

She stood the operation satisfactorily but there was no improvement in the neurological signs. From time to time, there were bouts of paraesthesiae and transient weakness of the right lower limb; it was thought that further infected emboli had lodged in the left hemisphere. The headache was relieved by the decompression, the spinal fluid pressure was normal and the fluid on 4th. April contained 35 mgm. protein and 32 cells (60 % neutrophils). Her general condition deteriorated steadily however ; restlessness and cyanosis were very

his temperature was normal. The respiratory rate was increased and he had a frequent loose cough with copious purulent sputum. He had two more generalised epileptic attacks, but three days before admission he had an attack in which the right side of the face, the right arm and the right leg were convulsed. Following this attack for the first time it was noticed that he had a definite weakness down the right side of the body. The hemiplegia progressed rapidly, he became comatose, the respiratory rate increased to 40/min., and purulent sputum welled out of his mouth when he coughed or retched. In this state he was admitted to the Radcliffe Infirmary. The pupils were dilated and inactive, the corneal reflexes were absent, and indeed all reflex functions were abolished. The pulse rate was 160/min., blood pressure 140/90.

He was clearly moribund, but it was felt that if he had a large abscess aspiration of its contents might tide him over the immediate crisis. Accordingly a left frontal burr hole was made within half an hour of admission. The brain was oedematous and the exploring needle encountered a small loculus containing 2-3 c.c. of greenish-yellow pus. This was clearly inadequate to account for his condition, and he died about half an hour later.

The autopsy revealed chronic bronchiectasis of the left lung with much purulent secretion in the whole bronchial system. There were multiple small abscesses in the brain, four or five presenting on the surface of each hemisphere and about ten times that number were counted in serial sections of the brain. Films from the pus aspirated during life showed gram-negative bacilli, some coliform and some fusiform. Cultures yielded a growth of bacillus fusiformis and a non-hemolytic streptococcus.

Case 4 . Chronic Left Fronto-Parietal Abscess (*Aspergillus*) due to Abscess of Left Lung. Extirpation of Cerebral Abscess. Recovery.

J.H., male aet. 37 (R.I.21249) was admitted on 12th. January, 1944, from the City General Hospital, Leicester, where he had been under treatment for peptic ulcer. Ten years before, he had had an operation for perforated duodenal ulcer, from which he recovered but he continued to have pain after meals, frequent vomiting, and he lost several stones in weight. These symptoms led to his admission to the Leicester hospital where it was found that he was suffering from pyloric stenosis, and in the early part of February, 1943, a partial gastrectomy was done under local anesthesia. His convalescence was complicated by some sepsis in the wound and by an abscess in the left lung (Fig. 30). Thereafter, his general condition improved, the abdominal wound healed, and he was discharged from hospital on 31st. March, 1943. Apart from a slight persistent cough, his general health continued to improve.

On 10th. June, 1943, he was sitting at table when quite suddenly his right arm began to jerk, he gave a cry and lost consciousness. He became cyanosed, frothed at the mouth, and the right arm and leg were jerking rhythmically. The fit lasted for about fifteen minutes and when he recovered consciousness it was found that he could not speak properly : in his wife's words, he "seemed to know what he wanted to say but could not get the words out" . There was also some weakness of the right arm and leg, but all of these abnormalities cleared up within an hour and after that he seemed to be perfectly normal. He returned to work as a shoe-factory operative in the first week of July, 1943.

FIGURE 32 overleaf

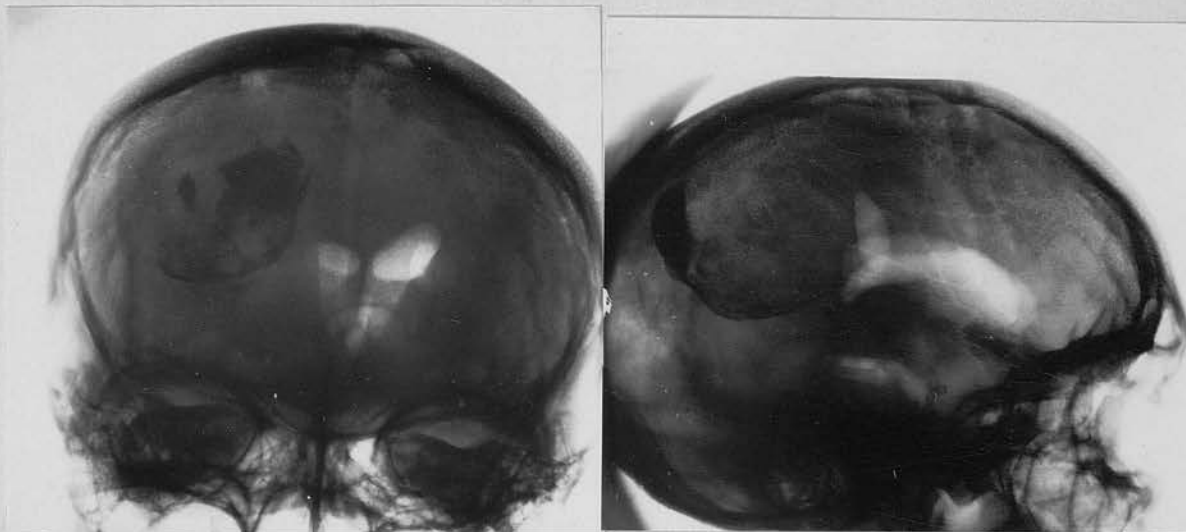
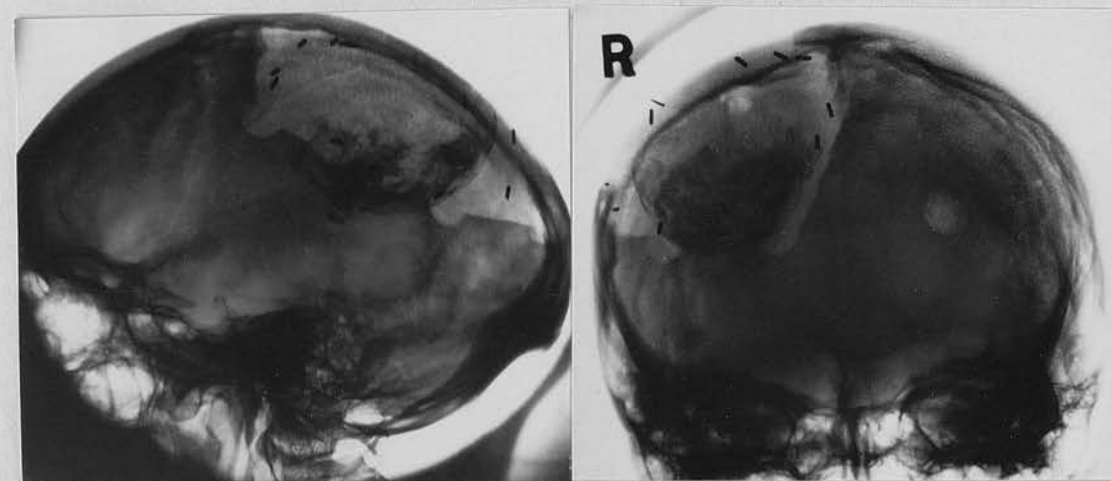
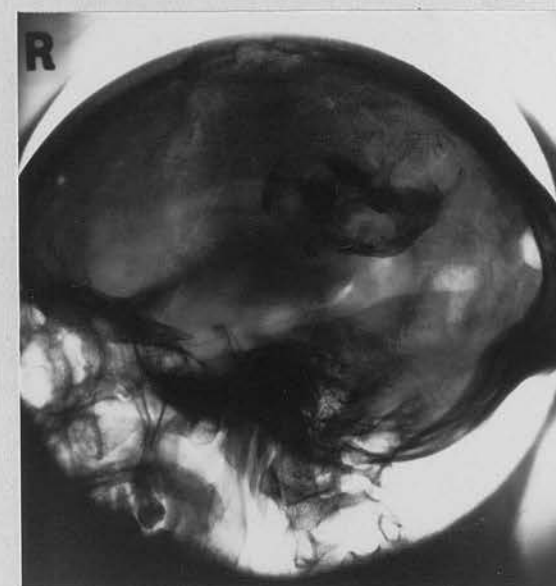
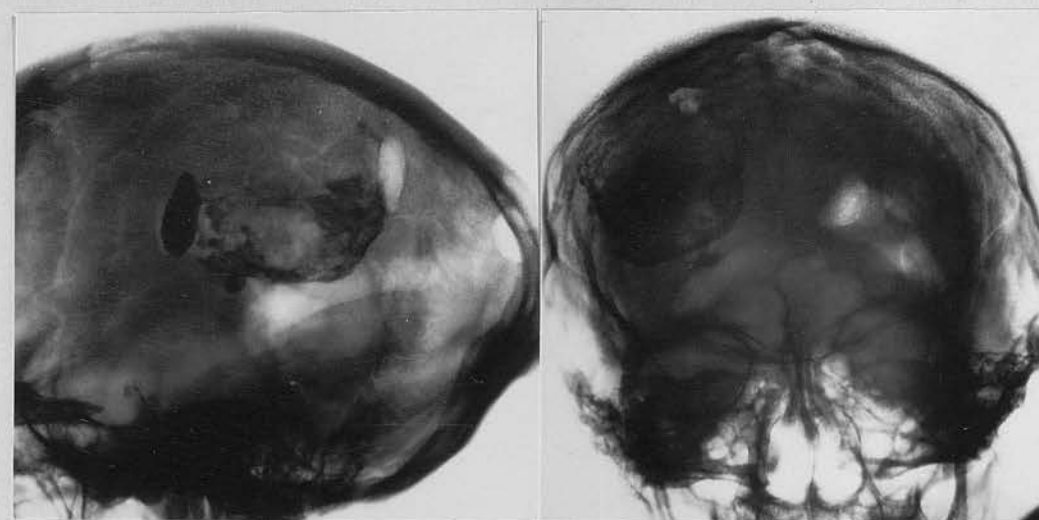


Figure 32. Case 5 - Ventriculogram and pyogram, 13:iii:42, showing displacement of ventricular system to the left side, and deformity of right lateral ventricle due to the large parietal abscess. Note the bubble of air in the abscess cavity in the lateral projections, from which the size of the abscess can be inferred.

The plates at the right were taken ten days after operation. They show the extent of the craniectomy in relation to the abscess, and the increase in the size of the abscess.



marked and frequent injections of morphia were required to relieve her distress before she died on 7th. April, 1942.

The autopsy revealed the characteristic visceral lesions of infective endocarditis. In the brain, there was a large encapsulated abscess (Fig.33) in the right parietal lobe which extended forward to the region of the Sylvian fissure. In the left hemisphere, Dr. Dorothy Russell demonstrated numerous embolic foci, for the most part of microscopic size, which doubtless accounted for the aphasia and sensori-motor signs in the right lower limb. Although these emboli were undoubtedly infected none of them had progressed to pus-formation.

FIGURE 33 overleaf

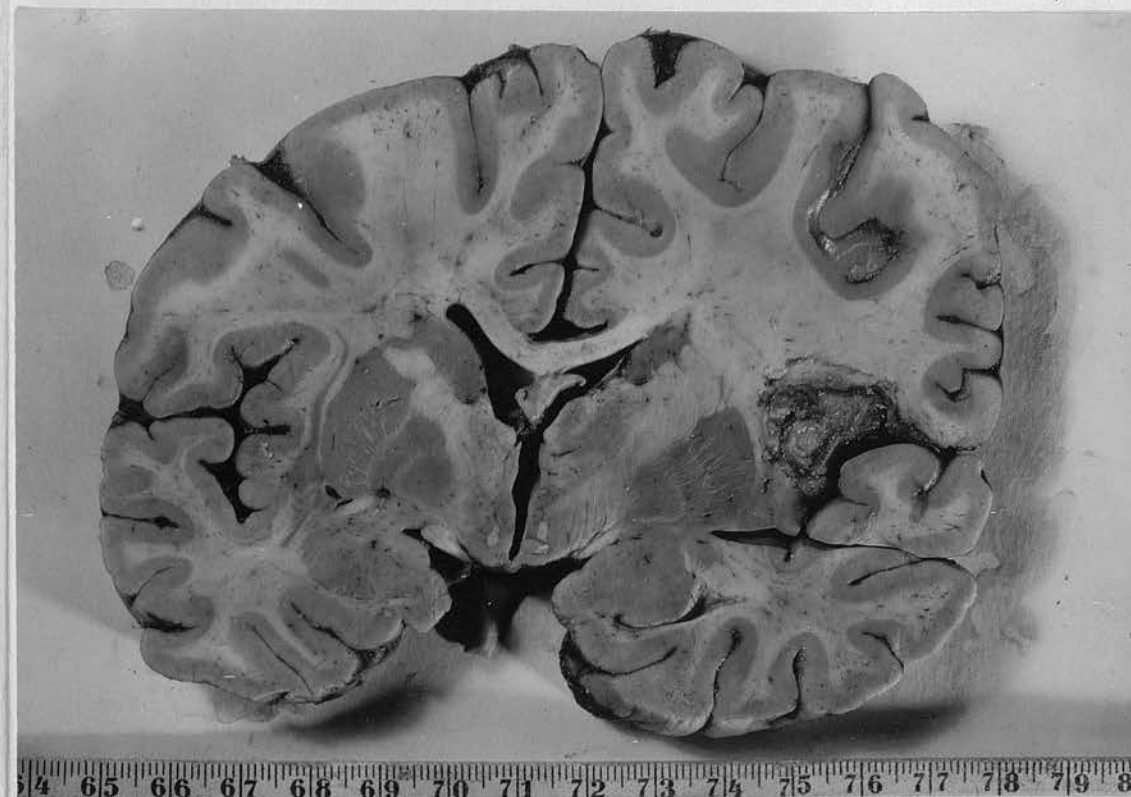


Fig. 33. Case 5 - Large right parietal abscess abutting onto wall of right lateral ventricle. The plate to the right shows the anterior pole of the abscess in the Sylvian fissure ; not the oedema of the white matter of the right hemisphere at this level.

Case 6. Metastatic Left Temporal Abscess (*Staphylococcus Aureus*). Suspected Origin in Staphylococcal Infection of Skin. Osteoplastic Decompression with Aspiration of Abscess. Osteitis of Bone Flap. Recovery.

W.S., male aet. 17 (R.I. 4725) was admitted to the Radcliffe Infirmary on 19th. November, 1938. He had been in good health until one month before admission when playing football he suddenly began to have an aching pain behind the eyes. He finished the game, but the pain got worse and assumed a throbbing character. He vomited two or three times, his neck felt stiff and it was painful to move his head from side to side. He also complained of giddiness and of "something wrong" with his eyesight which he later identified as double vision.

After a night's sleep, all of these symptoms were less marked and he carried on with his work although he had never been free of a dull frontal headache from the outset.

One week before admission, there was a recurrence of the severe headache, vomiting, giddiness and diplopia. These symptoms were so severe that he had to give up his work and go to bed. They persisted until admission.

On admission, he was in considerable distress with a severe frontal headache, but he was quite conscious and rational. Temperature and respirations were normal, but the pulse rate varied between 48 and 56/min. B.P. 140/80. A very slight nominal aphasia was demonstrated in formal tests but was not apparent in ordinary conversation. He was right-handed. The optic discs were engorged and there were several small hemorrhages in each fundus, with 2.5 D. swelling. The visual fields were full and the only other neurological abnormalities were slight weakness of the right side of the face and right upper limb,

with increase of the tendon reflexes in the right limbs, and a right plantar extensor response. There were no definite abnormalities in the X-rays of the skull. The spinal fluid pressure was over 300 m.m., and the fluid contained 65 mgm. protein, 128 cells (70 % lymphocytes).

The lesion was thought to be in the left frontal lobe, and the short history of increased intracranial pressure with the findings in the cerebrospinal fluid made an abscess a likely possibility. There was no history of ear disease or sinus infection but he had some acne pustules on his face, and he said that he had had mild "septic spots" in other parts of his body for several months. These were the only etiological factors which were ever discovered.

A ventriculogram (Fig. 34) on 22nd. November, 1938, indicated a lesion in the anterior part of the left temporal lobe. An osteoplastic flap was reflected on the same day and through the unopened dura a brain needle was inserted into the abscess and 12 c.c. of pus were aspirated. No capsule could be felt and it was decided to leave him with the bony decompression, pending the formation of a sufficiently thick capsule to allow the abscess to be extirpated. Staphylococcus aureus was cultured from the pus.

After operation he had no further headache, the slight aphasia and right hemiparesis cleared up, and the papilloedema began to subside. Ten days after operation, the anterior limb of the scalp incision became inflamed and began to discharge staphylococcal pus. This discharge continued, and by 18th. January, 1939, (eight weeks after operation), there were radiographic signs of infection of the bone flap (Fig. 34), so on this day the scalp flap was re-elevated and the anterior third of the bone flap was removed; the remainder of it looked healthy. The wound healed

FIGURE 34 overleaf

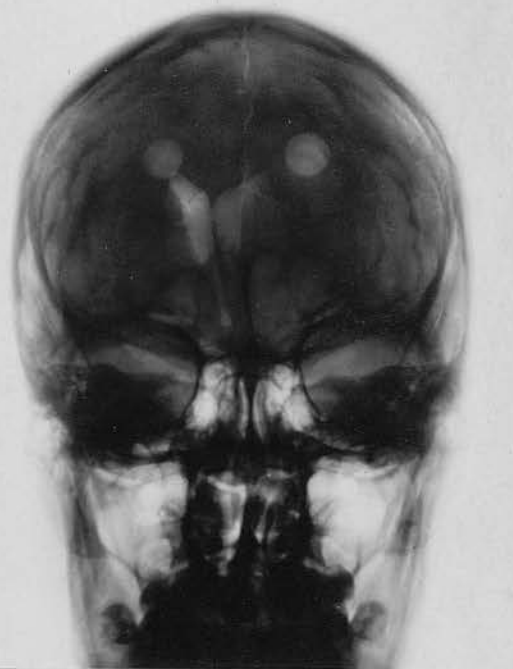
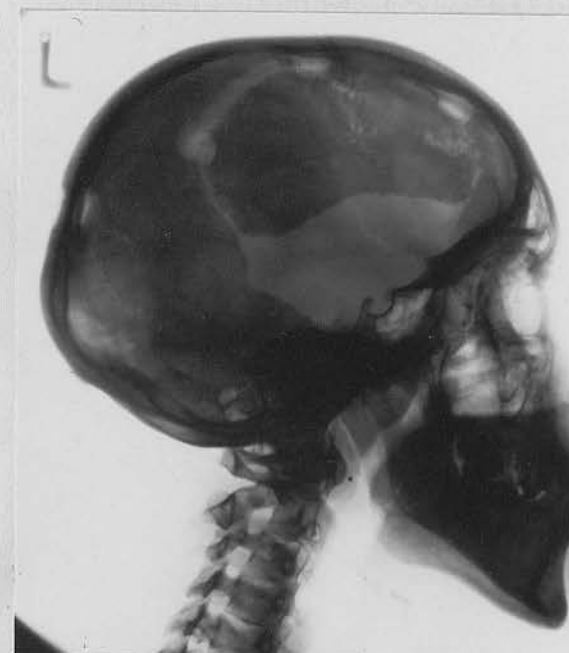


Fig. 34. Case 6 - ventriculogram on 22d. November. Note displacement of ventricular system to right side, and in the lateral and axial projections, the obliteration of the tip of the left temporal horn. This is an unusual finding in temporal lobe abscesses, as most of them, due to mastoid disease, are situated farther back in the temporal lobe and it is rare to visualise the temporal horn at all or indeed any part of the lateral ventricle on the side of the lesion.

The plate to the right shows evidence of infection of the antero-superior part of the bone flap. It was taken eight weeks after the operation.



and his general and neurological improvement was maintained. By 16th. March, 1939, the spinal fluid had returned to normal (40 mgm. protein, 2 cells), the pressure was normal, the papilloedema had subsided and he was free from symptoms and signs. He returned to work in June, 1939, and has remained well since then.

Comment : This was a small abscess, and the great increase in intracranial pressure must have been due to oedema around the abscess rather than to the size of the abscess itself. The effect of the aspiration and decompression must have been to relieve the oedema to such an extent that more blood could be brought to the infected zone, and thus allow the natural defences to operate against the infecting organisms. It is unusual for a single aspiration to have such an effect, and had there been any recurrence of symptoms or signs, further aspirations would have been done with a view to ultimate removal of the abscess.

Case 7. Multiple Right Parieto-Occipital Abscesses of Undetermined Etiology. Drainage by Tube, with Subsequent Marsupialisation. Formation and Excision of Fungus Cerebri containing Multilocular Abscess. Recovery.

S.H., male aet. 28 (R.I. 2019) was admitted on 20th. May, 1938. Except for uncomplicated scarlet fever at the age of three years he had had no illnesses until 15th. April, 1938, five weeks before admission. On this day he was out for a country walk when he suddenly "began to see coloured lights dancing about " in his left visual field. They were probably in the lower quadrant as he said that they "seemed to be between 7 and 8 o'clock" . This sensation persisted for two or three minutes , then he carried on with his walk. For the next two or three days he had no complaints but his mother thought that he was depressed and lacking in his usual vitality. He then told his mother that he could only see things directly in front of him and that everything looked misty. He was sent to an eye specialist who found no abnormality. During the next fortnight the only striking development was that the mental depression became much more marked and he was quite "unlike his usual cheery self" .

Three weeks before admission, he began to complain of headache chiefly in the right side of the head and behind the right eye. On several occasions he vomited and he had to give up his work and go to bed two weeks before admission. In the last two or three days he had been very sleepy and at times he was difficult to rouse.

On admission, he was wasted and ill-looking, and so drowsy that little co-operation could be got from him. After a rectal infusion of magnesium sulphate, he brightened up and it was possible to carry out a fairly complete examination. Both optic fundi were normal and

he could read at least Jaeger 8 with each eye. There was a complete left homonymous hemianopia, and he was able to describe the development of this defect : he said that he had had several of the attacks of flashing lights in the left visual field, and after the first one on 15th. April he was aware of a dark patch in the lower left field which persisted and became more extensive after each attack.

Both eyelids were slightly drooped and there was a defect in upward movement of the eyeballs, but the pupils and ocular movements otherwise were normal. He had a slight left hemiparesis affecting face, arm and leg equally ; the tendon reflexes were present and equal, but the left plantar response was extensor whereas the right was flexor. Perception of light touch and pin-prick was slightly impaired down the left side of the body, and there was a well-marked defect of postural sensibility, two-point discrimination and thermal sense in the left limbs. X-rays of the skull and chest were normal.

On 22nd. May, he was much more drowsy and vomited twice. The temperature was 99 degrees, pulse rate 60/min., and respirations were slow(12/min.) punctuated by periods of apnoea. He was slightly cyanosed. The only striking neurological abnormality which developed under observation was that the right pupil had become dilated and immobile.

There was clearly an expanding lesion in the posterior part of the right cerebral hemisphere. In the absence of any history of infection, a rapidly-growing (or cystic) neoplasm was thought to be more likely than an abscess. A lumbar puncture was done, and despite the stupor the pressure was only 150 m.m. (see Case 9). The fluid contained 70 mgm. protein and 31 cells (54 % lymphocytes), but this analysis was not

available until after the diagnosis had been made, as immediately after the lumbar puncture a ventriculogram was done and in needling for the right ventricle through a parietal burr-hole an abscess was encountered at a depth of 5 cm. From this some 15 c.c. of pus were aspirated. The burr hole was enlarged down toward the temporal region and an exploring needle here encountered pus 1.5 cm. from the surface. A firm wide-bore rubber tube was inserted into the abscess cavity and a further 8.c.c. of pus allowed to drain off. The tube was fixed in position and the scalp wound sutured around it.

At the end of the operation his colour was better, respirations were improved, but he remained very drowsy. The right pupil was still dilated and fixed. On the following day he swallowed well (84 oz.) but there was no improvement in the stupor. There was no appreciable discharge from the drainage tube.

On 24th. May, he could not be roused at all, and it was clear that something more would have to be done. As before, the spinal fluid pressure was not high, 140 m.m. A cruciate incision was made over the temporo-parieto-occipital region with its centre at the opening for the drainage tube, and the four angled skin flaps were turned back and sutured to the scalp. A circle of dura 4 cm. in diameter with its centre at the drainage opening was excised. The brain began to herniate through this opening and a circle of cortex was quickly removed. The wall of the abscess was found at a depth of 2 cm. and it was incised. About 10 c.c. of pus were sucked out. The wall of the abscess was about 0.2 cm. thick but it was impossible to stitch it up to the dura until it had been dissected away from the surrounding white

matter. When it had thus been partially mobilised, the opening into the abscess was enlarged and the inferior lip of the aperture was sutured to the dural edge. A drainage tube was left in the cavity, and the wound was left open.

As was expected a fungus cerebri developed. It was covered by clean granulation tissue at the end of a week, and there was practically no discharge from the cavity of the abscess. Daily or twice-daily lumbar punctures were done, the pressure varying between 170 mm. and 240 mm. The removal of 10-15 c.c. of fluid slackened the tension of the fungus but did not diminish its size appreciably. The fluid was always clear and colourless, the protein content varying from 70 mgm. to 125 mgm., and there were usually 6-8 lymphocytes.

His general condition improved greatly. He was usually sleepy in the morning, but later in the day he was alert and took a keen interest in current events. He had a voracious appetite, and that he was using the food was shown by the fact that the wasting became less marked and his mother said that "he was looking more like himself".

As he became more alert, it was apparent that the dilatation of the right pupil was only part of a complete third nerve palsy. The left homonymous hemianopia persisted, as did the sensori-motor affection of the left side.

During the first two weeks of June, the fungus increased in size until it was about the size of half a tennis ball projecting above the level of the scalp. It then began to recede, and suddenly on 18th. June it collapsed and became markedly indrawn. It remained so for three days but when the dressing was done on 21st. June it was larger

than ever and more tense. For the next five or six days, lumbar punctures were done at 12-hourly intervals, 30-50 c.c. of fluid being removed on each occasion, to slacken the tension in the fungus. His general condition continued to improve, and he was allowed to get up and walk around. By 15th. July, the fungus was just flush with the skull, showing no tendency to bulge. He seemed to be very well in himself, but he found the hemianopia and third nerve palsy to be very troublesome. Covering one eye did not much help matters.

On 19th. July, the fungus was again tense and he vomited twice. The spinal fluid contained only one cell, but 190 mgm. protein. Four days later, the fluid was slightly turbid and contained 167 cells : this coincided with an increase in headache, a rise of temperature to 101 degrees, and of the pulse rate to 110/min. His appetite failed and he was a little lethargic.

On 27th. July, he was very drowsy, the fungus was tense, and the spinal fluid pressure was 280 mm. The fluid was clear and colourless, containing 120 mgm. protein and 63 cells. Some specimens of fluid were slightly yellow, although all were perfectly clear. The bilateral ptosis had recurred, upward movement of the eyes was defective, and the left hemiparesis was more profound. In an attempt to determine the size and extent of the abscess, a ventriculogram was done but this only succeeded in filling the left ventricle, which was profoundly displaced to the left side. In needling for the right ventricle a small collection of pus was encountered in the depths of the right parietal lobe. He was taken to the theatre and the scalp flaps were re-elevated and extended to expose the posterior half of the

skull vault on the right side. The bone was removed posteriorly to the level of the transverse sinus, and medially to the margin of the sagittal sinus. The fungus projected above the level of the dura to the size of a tennis ball, but the exposed dura was not unduly tense. It was opened around the base of the fungus, and in the posterior part of the parietal lobe and the anterior part of the occipital lobe the abscess wall could be felt about 1 cm. below the surface of the cortex. A wide cortical excision was made, and the honey-comb of a multilocular abscess was exposed and sucked out. There were eight or nine separate and sizeable loculi which did not appear to communicate with each other, and there were numerous smaller pockets which could not be counted. Gauze wicks were left in the larger cavities, and the enlarged wound left open to fungate.

(This operation was done under local anesthesia in the prone position to afford access to the posterior half of the hemisphere. Afterwards, he had a complete paralysis of the right upper limb, with absent tendon reflexes and a slight sensory impairment from the 4th. cervical to the 2nd. thoracic segments. This was due to pressure of the shoulder rest in the right axilla, and it took about six weeks for the paralysis to clear up.)

Again there was improvement; it continued for two weeks and then he began to lose ground. As on previous occasions this was shown by depression, anorexia, lack of interest in his surroundings, and finally by progressive stupor. The fungus increased in size and the spinal fluid pressure was constantly above 200 mm. The protein content varied between 200 mgm. and 400 mgm., but there were rarely more than 4-6 cells. The withdrawal of 40-50 c.c. of spinal fluid had no

appreciable effect on the size of the fungus, although it would of course slacken the tension.

On 20th. August, he was operated on for the fourth and last time. The whole fungus was excised and further loculi were broken down and sucked out. The cavity was for the first time converted into a single one with smooth firm walls. A bulge was seen in the floor of this cavity, and thinking that this was yet another loculus deep to the main mass, we made an incision into it. It proved to be the vestibule of the lateral ventricle. Fortunately its wall abutting on the abscess was thick enough to take stitches, and a water-tight closure could be made. The abscess cavity was lightly packed with ribbon gauze soaked in azochloramide and the wound was left open.

From that time his progress was uninterrupted. The cavity gradually filled out and became lined with clean granulation tissue. It was two months before it was completely epithelialised and finally healed, and it was markedly indrawn through the defect in the skull. The protein content of the spinal fluid was 50 mgm. on 7th. October, and there were 9 lymphocytes. By 3rd. November, it had returned to normal (30 mgm.protein, 2 cells).

His general condition improved : he put on weight to his normal level, the depression lifted , and he had no symptoms except those referable to his eyesight. On analysis, he had a complete left homonymous hemianopia splitting the fixation point, but the acuity in each eye was 6/6, J.1. The right third nerve palsy had cleared up except for a slight weakness of the medial rectus, and formal diplopia tests suggested that there was also a little weakness of the superior

oblique muscle. These defects seemed to be insufficient to account for his complaints and it was found to be a disturbance of spatial orientation. He would get lost in the ward corridor and not be able to find his way back to his bed, or if taken for a simple walk in the hospital grounds, he would be unable to find his way back although there were many prominent landmarks to guide him. He said himself that his "visual memory had gone" - he could not remember what his friends and some of his relatives looked like, and he was unable to picture the journey from his own home to his work. These symptoms were still present when he was discharged on 9th. November, 1938, but there were no other neurological abnormalities.

He returned to work in April, 1939, and has remained well since then, although he still has great difficulty in getting adjusted to the hemianopia and diplopia. The defect in spatial orientation persisted for several months, and four years after operation he said that he would still get lost in strange surroundings if he did not pay particular attention.

Comment: This case was dealt with before our ideas about treatment were clearly formulated. Although the ultimate result was probably as good as could have been obtained by any method of treatment, the open drainage method entailed his being in hospital for nearly six months, having hundreds of lumbar punctures and dressings done, and four major operative procedures. We feel now that this case would have been more efficiently dealt with by decompression and ultimate extirpation, with closure of the wound.

Case 8. Left Temporal Abscess (pneumococcal) following Spontaneous Resolution of Left Otitis Media. Repeated Aspiration and Eventual Removal of Multilocular Abscess. Recovery.

K.C., male aet. 8 (R.I.20226) was admitted on 26th. December, 1941. He had always been in good health until the last week of September, 1941, when he complained of acute pain in the left ear, and this was followed after a day or so by the discharge of some thick yellow pus from the ear. The discharge lasted for a week, at the end of which time he had no pain or any other symptoms, and he seemed to be in his usual good health. From that time, however, he complained of occasional headache, especially on waking in the morning, and on two or three occasions he was sent home from school because of the headache.

On the 14th. December the headache was so severe that his mother did not send him to school, and because of frequent retching and vomiting he was kept in bed. These symptoms led to his admission to the Bedford County Hospital on 20th. December, 1941. In the six days before transfer to the Radcliffe Infirmary, the headache and vomiting persisted, he became a little drowsy and was occasionally incontinent of urine. The diagnosis of tuberculous meningitis was suggested but a lumbar puncture revealed clear, colourless fluid under a pressure of 300 mm., and the fluid contained 30 mgm. protein, 5 lymphocytes, 720 mgm. chlorides. Cultures were sterile.

On admission to the Radcliffe Infirmary, he was conscious but drowsy and complaining of headache. It was difficult to get him to speak, but when he did it was clear that he had an expressive aphasia. There was bilateral papilloedema, 2 D., and a complete right homonymous hemianopia. There was a partial sixth nerve palsy on the right side producing a slight squint. There was profound

weakness of the right side of the face, so marked that it resembled that seen in nuclear or lower motor neurone lesions, but there was also marked weakness of the right upper limb and to a less extent of the right lower limb. The tendon reflexes were diminished, but equally so. The right abdominal reflexes were absent whereas the left were present. Both plantar responses were flexor. There was no definite sensory disturbance. The right ear drum was normal ; the left was intact but there was a small scar at the site of the perforation three months earlier. There was no mastoid tenderness and no clinical evidence of meningitis.

The evidence thus favoured an expanding lesion in the left temporal lobe, and despite the quiescence of the aural infection and the nearly normal spinal fluid, abscess seemed the most likely possibility. Through a left parietal burr hole, the abscess was encountered in the temporal lobe and 25 c.c. of pus were aspirated, and 2 c.c. thorotrast instilled into the cavity. This was done on 27th. December (Fig. 35) , and there was immediate improvement : he was more alert, said that his headache had gone , and that he was very hungry. The right hemiparesis improved, but there was no change in the hemianopia. Two days later, he again became very drowsy, and as the skiagrams had shown a large abscess (Fig. 35) another burr hole was made just above the pinna to afford more direct access for repeated aspirations. On this occasion, 75 c.c. of pus were aspirated and subsequent X-rays showed (Fig. 35) that the cavity had collapsed to some extent. Again there was a dramatic improvement in his general condition of alertness, but because of recurring stupor and headache, it was necessary to repeat the aspirations on 9th. January, 1942 (80c.c.), and

FIGURE 35 overleaf

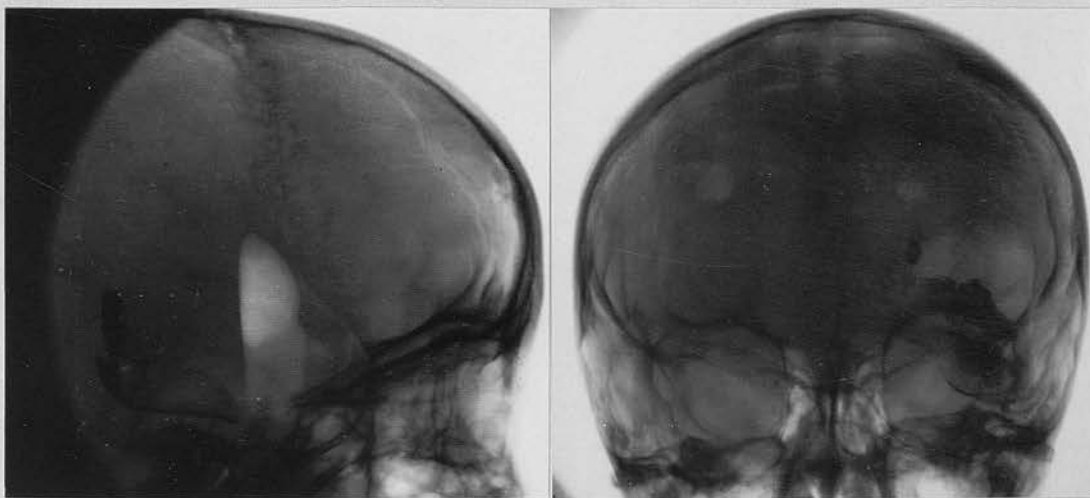
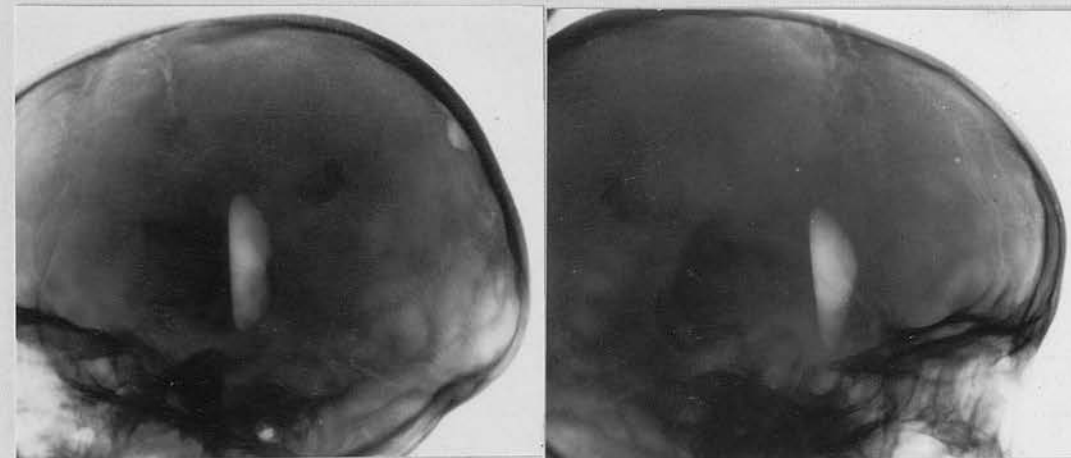


Fig. 35. Case 8 - Pyrogram after first aspiration. Note air in abscess cavity, and wavy outline of walls showing that the tension in the cavity is low.



Pyrogram two days later (29:xii:41) showing increase in size of abscess, and more "roundness" suggesting tension within its cavity. Its antero-posterior extent can be judged from the air bubble, as the plate on the right was taken with the brow uppermost, while that on the left was taken with the occiput uppermost.

The three pictures to the right were taken immediately after aspiration on 29:xii:41. Compared with the ones at the upper right, they show that the air has been aspirated and that the whole abscess cavity is considerably smaller.

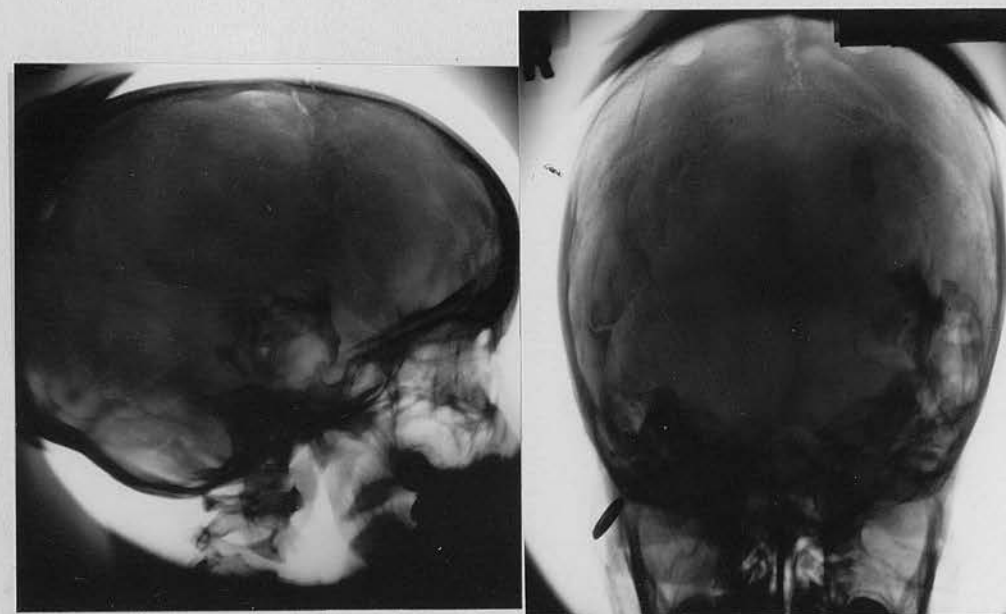
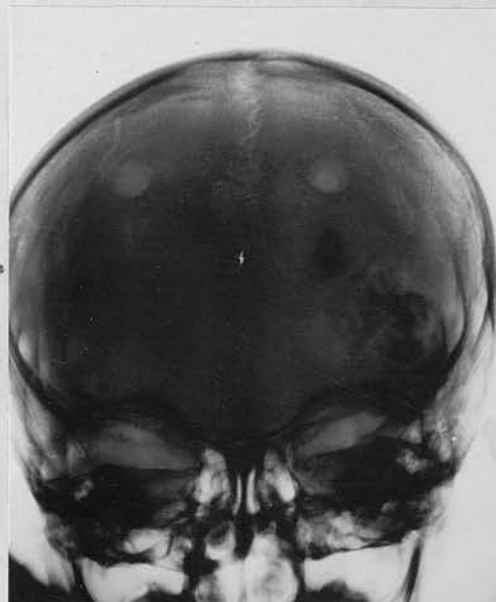


Fig. 36 overleaf.

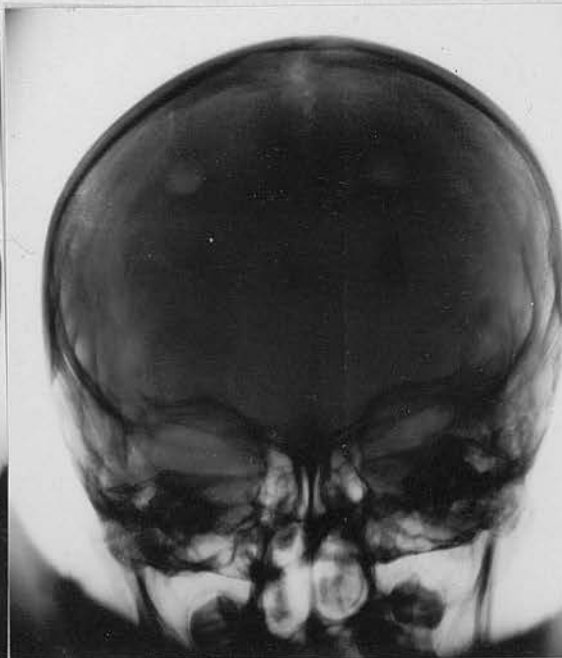
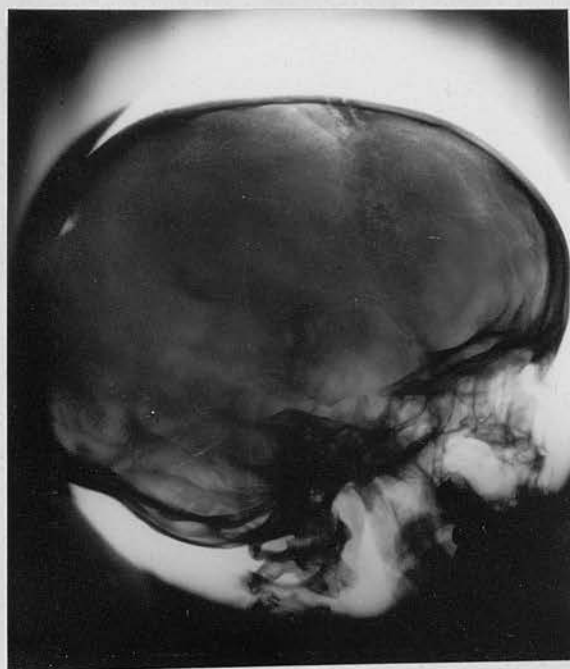
Case 8.



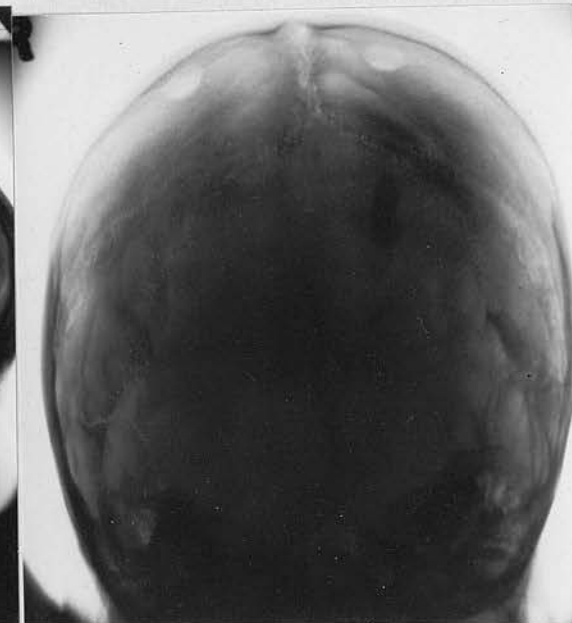
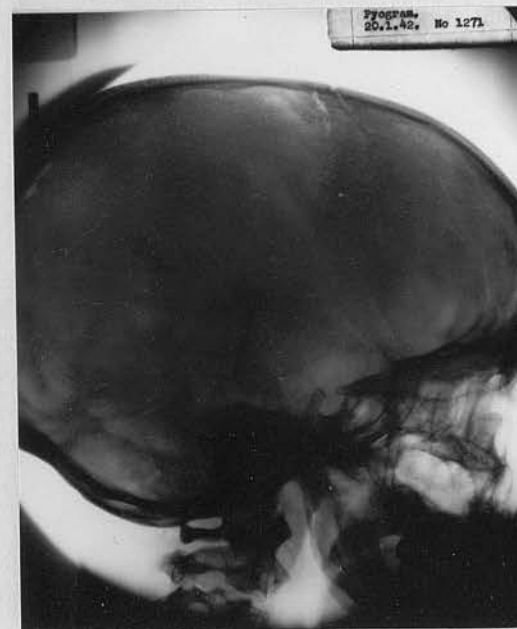
Fig. 36. Case 8. Pyogram 7th. January, 1942, after first aspiration



Pyogram 13th. January, showing increase in size of abscess.



Pyogram 13th. January, after aspiration : compare with the plates at upper right before aspiration.



Pyogram 20th. January, showing further increase in tension after last aspiration a week earlier (see plates at left.)

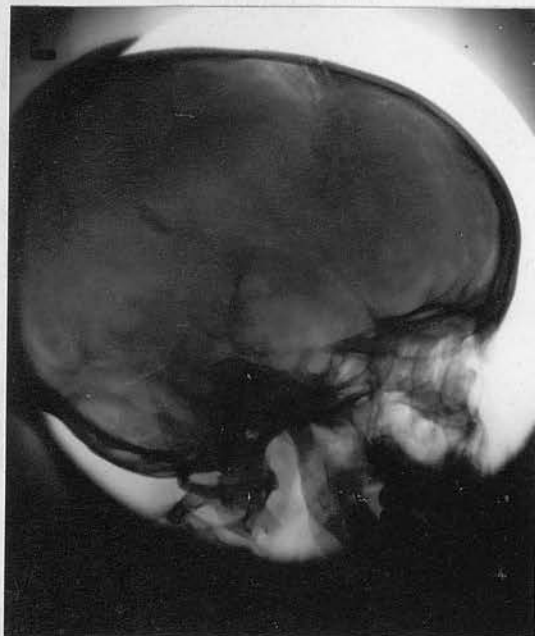
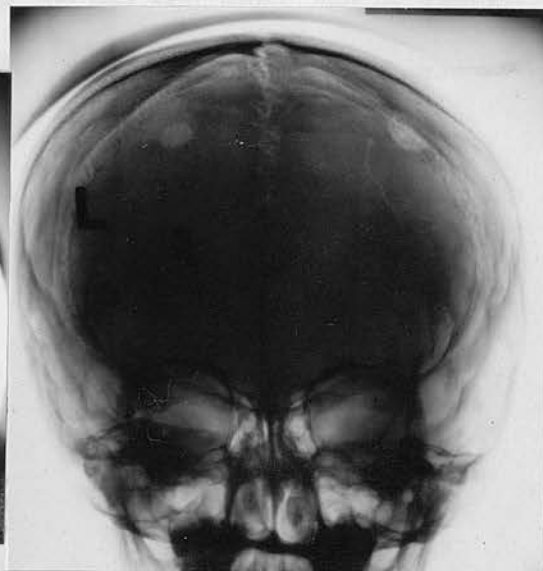
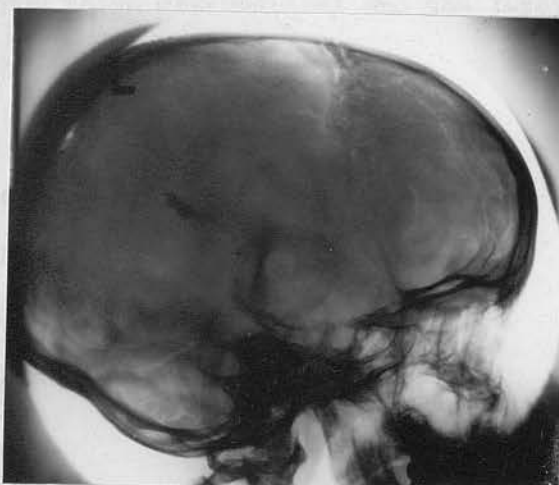


Fig. 36a. Case 8 - pyrogram 27:i:42 and 23:ii:42, showing variations in tension of wall of abscess with repeated aspirations.



Pyrogram 3:iii:42, immediately prior to operation .

13th. January (72 c.c.) . He then began to improve : headache lessened, there was no vomiting, the aphasia was less marked, and the right hemiparesis was hardly demonstrable. There was no improvement in the papilloedema however, and the right hemianopia persisted. The spinal fluid pressure, taken on many occasions during this period, varied between 230 and 300 mm. The protein content varied between 80 mgm. and 120 mgm. The cell count ranged from 8 to 20, mostly lymphocytes.

The papilloedema increased and hemorrhages appeared despite the fact that the child was getting up and about, complained of no headache and had no definite neurological signs except the right homonymous hemianopia. The hemiparesis and aphasia had almost entirely disappeared as had the sixth nerve palsy. The spinal fluid pressure continued to vary between 250 mm. and 300 mm. and there was no doubt from these observations and the X-ray appearances (Fig. 36) that the abscess was under considerable tension.

It was decided to try to sterilise it with penicillin injections, so on 7th. February, and again on 9th. and 10th. February, the abscess was aspirated and penicillin injected. On the first occasion 12 c.c. of pus were removed ; on the second, 10 c.c.; and on the third, 35 c.c. On each occasions , 5 c.c. penicillin solution were injected into the abscess. At the last aspiration, there was some difficulty in getting the needle into the cavity and it was felt that the abscess probably had a sufficiently thick capsule to render extirpation possible. The penicillin injections were stopped. His general condition remained stationary but the papilloedema increased slowly and it seemed that the time had come for the removal of the

abscess.

On 3rd. March, 1942, a left lateral osteoplastic flap was reflected. The dura was very tense and an attempt was made to lower the pressure by tapping the right ventricle, but it could not be found so a sharp needle was passed into the abscess and 30 c.c. of pus were aspirated. This slackened the dura sufficiently to allow it to be opened easily. The abscess could be seen coming near to the surface in the middle three fifths of the temporal lobe. The cortex was thinned to less than one centimeter over the abscess, and the capsule of the abscess was exposed by removal of this thinned cortex in an ellipse 3.5 cm. x 2 cm. The capsule was quite firm and the dissection from the surrounding white matter was straight-forward. There were two major loculi : the larger and more superficial one, and a smaller deep loculus with thin walls which abutted on to the temporal horn of the ventricle. The larger loculus was tethered to the dura over the tegmen tympani by a short stalk, and when this was divided the whole mass lifted out in toto. The removal of the deeper loculus entailed the removal of the lateral wall of the temporal horn, so that the ventricle was thrown into wide communication with the large cavity in the temporal lobe from which the abscess had been removed.

The abscess mass measured 7 cm. x 6 cm. x 5 cm. On section, the larger loculus was found to contain four smaller loculi filled with thick grey pus. The smaller loculus had a thinner wall (0.2 cm.) and it contained creamy yellow pus. There were many gram negative diplococci in the superficial loculi (which presumably were the ones treated with penicillin) but none in the deeper loculus.

Convalescence was uneventful. The wound healed normally, and

he was getting up and about from the fifth post-operative day. The spinal fluid pressure was normal from the time of operation, and the papilloedema subsided. The homonymous hemianopia remained complete, except for macular sparing, and his visual acuity was 6/6, Jaeger 1 in each eye. By 2nd. April, there was no aphasia nor any demonstrable neurological abnormality except for the hemianopia. The spinal fluid however took six weeks to revert to normal : this has been observed in other cases in which communication is established between the operation cavity and the ventricle.

He has remained well, and two years after operation his school-master reported that he had at least maintained his previous average. There were no neurological defects except the right homonymous hemianopia to which he seemed to have made a satisfactory adjustment.

Case 9. Right Temporal Abscess due to Middle Ear Disease. Repeated Aspiration, Decompression, Eventual Removal of Abscess. Recovery.

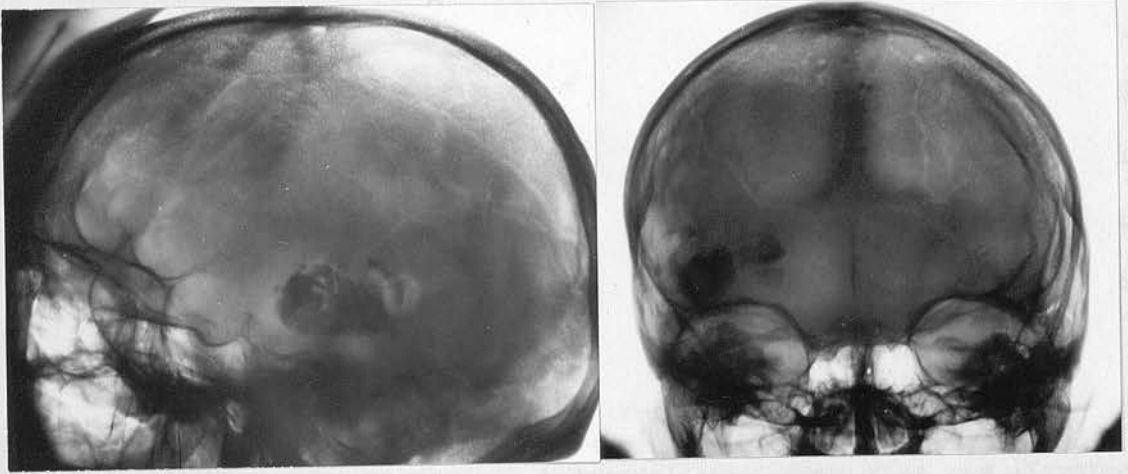
B.J., male aet.48 (R.I. 19215) was admitted on 4th. November, 1941. He had been in good health until six weeks before admission when he complained of pain in the right ear and the right side of the head. On the third day of this illness the pain was so severe that he had to go to bed ; he vomited several times, and it was found that there was a slight elevation of temperature. On the fifth day, a yellow purulent discharge appeared in the right external auditory meatus, and his doctor found that the tympanic membrane had ruptured. There was no dramatic relief of the pain, but five days after the discharge began, the pain abated and by the end of three weeks it had ceased entirely. The aural discharge persisted for three days and never recurred. Thus at the end of three weeks he seemed to have recovered completely and he returned to work and his normal activities.

Two days later, while at a Rugby match, he had a feeling of nausea which made him go home and to bed. On the following day he had a severe headache and vomited several times. He began to hiccough, and this persisted until the time of his admission. He was delirious and confused at times, but at other times appeared to be quite rational, complaining of severe pain in the back of the head and stiffness of the neck. He became progressively more drowsy although at no time had it been impossible to rouse him. There had been no epileptic phenomena, no visual symptoms, and he had not been aware of any particular weakness of his limbs.

On admission, he was a well-nourished man, drowsy, but capable of being roused to co-operate in a full examination. He was slightly confused and disorientated, and was obviously distressed by severe headache and frequent hiccoughing. There was some stiffness of the neck. The temperature was 97.8 degrees, pulse rate 68/min., respiration rate 16/min., Blood pressure 120/80. The positive neurological signs were engorgement of both optic discs, without measurable swelling ; an upper quadrant defect in the left homonymous field of vision ; slight bilateral ptosis ; slight weakness of the left side of the face and of the left upper limb ; diminution of the left abdominal reflexes, and a left plantar extensor response. On lumbar puncture the pressure was 200 mm. ; the fluid contained 50 mgm. protein and no cells. X-rays of the skull showed some relative opacity of the right mastoid region.

The diagnosis of right temporal abscess was fairly clear, and on the day after admission he was so drowsy that it was decided to aspirate the abscess (5th. November, 1941). A burr hole was made in the right squamous temporal bone and at a depth of 2 cm. the exploring cannula was felt to slip into a cavity from which 17 c.c. of greenish yellow pus were aspirated, (Fig. 37). The pus contained pneumococci in pure culture.

The effect of this aspiration was to relieve the headache and hiccough, and to make the patient much more alert. There was no significant change in the neurological signs, except that the ptosis became less marked making him look much more wide-awake. The general improvement was maintained for seven or eight days, when the headache, hiccough, and drowsiness began to recur. By 17th. November, 1941, he was extremely



Pyrogram on 5th. November, 1941, after initial aspiration.



13th. November, 1941



17th. November, 1941

Fig. 37. Case 9 - Pyrograms showing site of abscess, and variations in size. Note relation to middle cranial fossa and burr hole used for aspiration.

drowsy and the weakness of the left side of the face and of the left upper limb was very marked. A further 25 c.c. of pus were aspirated from the right temporal lobe. Again he became much more alert, and the left-sided weakness less marked. These effects persisted for three days, when it was noticed that he was sleeping more than he should and by 24th. November, 1941, he was in a moderately deep coma. Aspiration on this occasion recovered 12 c.c. of pus, and again he roused to the level of responding verbally, swallowing, and moving about in bed. This improvement was short-lived and by 27th. November he was again deeply comatose. On this occasion an attempt to aspirate the abscess failed, and it was felt that a decompression was necessary. A lateral craniotomy flap was elevated, and, rather surprisingly, the dura was found to be quite slack, so much so that it was not considered necessary to open it. A blunt brain needle was used to probe the abscess, and it was found to be in the usual place, i.e., the inferior part of the temporal lobe, and it had a capsule which was thick enough to be appreciated easily by the exploring needle. The capsule was pierced, and a further 8 c.c. of pus were aspirated. The bone flap was replaced and the wound closed.

This operation was completed at 9 p.m. By 11 p.m., although pulse, respiration and temperature were normal, he was more deeply comatose, having lost the corneal reflexes, all tendon reflexes, and he was unresponsive to painful stimuli. Despite this abolition of reflex function the spinal fluid pressure was only 140 mm., and the pressure in the left lateral ventricle was atmospheric. It was felt that the coma was probably due to odema of the temporal lobe, but the administration of intravenous hypertonic solutions brought about no improvement. It seemed that his only chance rested in the extirpation of the abscess.

The bone flap was re-elevated and when the dura was opened, the whole of the temporal lobe was found to be very oedematous. The abscess was dissected out intact, it being attached to the dura over the tegmen tympani by a short sessile stalk some 0.75 cm. in diameter. The medial surface of the abscess formed the lateral wall of the temporal horn of the ventricle, and the extirpation thus entailed making a wide opening into the ventricle. The cavity was irrigated with peroxide and proflavine and was lightly dusted with sulphanilamide powder. The bone flap was replaced and the wound closed without drainage.

The wound healed uneventfully. The spinal fluid had reverted to normal (35 mgm. protein, 2 cells) by 15th. December, 1941, and there were no abnormal neurological signs, except an incomplete left upper quadrant defect in the visual field. Doubtless this was the result of excision of the relevant part of the optic radiation during the extirpation of the abscess. This man returned to work in February, 1942, and has remained in good health since then.

Case 10. Staphylococcal Abscess of Right Temporal Lobe due to Chronic Mastoiditis. Mild Meningitis. Radical Mastoidectomy. Repeated Aspiration of Brain Abscess. Recovery.

E.H., female aet. 56 (R.I. 23850) was admitted on 11th. June, 1942.

She had had intermittent discharge from the right ear since childhood but was in good general health until 28th. May, 1942, when she began to suffer from acute pain in the right ear. For the next three days the pain continued and was associated with severe generalised headache and vomiting.

By 2nd. June, she became drowsy, irrational and incontinent. There was marked stiffness of the neck and Kernigism, and a lumbar puncture revealed turbid fluid under a pressure of 180 mm. There was no evidence of sinus occlusion on Queckenstedt's test; the fluid contained 1500 cells, most of which were polymorphs, and 120 mgm. protein. It was sterile on culture. She was put on M & B 693, and a radical mastoidectomy was done on this day : there was extensive disease of the mastoid, pus in the antrum, and erosion of the tegmen tympani.

The drowsiness persisted after the mastoid operation. By 9th. June it was possible to demonstrate a left homonymous hemianopia, and a slight left hemiparesis, maximal in the face and upper limb. There was no papilloedema. The spinal fluid pressure was 180 mm., the fluid was clear and colourless, and it contained 65 mgm. protein, 11 cells (10 of which were lymphocytes). The diagnosis of right temporal abscess was made.

By 13th. June, she was much more drowsy, almost to the point of not responding at all. Pulse 80/min., temperature 99 degrees, respiration 20/min., blood pressure 110/70. The left heiplegia appeared to be nearly

complete. A burr hole was made over the right ear ; the dura was tense and when it was incised the underlying brain began to protrude through the opening. A blunt brain needle was introduced, and at a depth of 1 c.m. from the surface it was felt to enter a cavity from which 18 c.c. of thick greenish pus were aspirated. One c.c. of thorotrast was put into the cavity and the scalp wound closed with two layers of stitches. An X-ray taken immediately afterwards showed the abscess outlined by thorotrast (Fig. 37). *Staphylococcus aureus* was cultured from the pus.

There was immediate improvement : she became much more alert and rational, the left hemiplegia largely cleared up, and the lower left homonymous field of vision expanded, although there was still an upper quadrant defect. Improvement continued for two weeks, but on 27th. June, she again complained of some headache and the drowsiness and left-sided weakness recurred. These signs became more pronounced during the next three days and X-rays showed that the abscess had increased in size. Accordingly a Greenfield lumbar puncture needle was inserted through the scalp over the burr hole and passed into the abscess from which 25 c.c. of thick pus were aspirated.

The effect was even more striking than after the first aspiration : she woke up immediately, seemed to be quite rational, and the weakness of the left limbs was barely perceptible, although the left facial weakness was still obvious. By the following day she was free from symptoms and it was not possible to demonstrate any neurological abnormality except a slight defect in the left upper field of vision. Her relatives were ardent Christian Scientists and they were so impressed by her recovery that they refused further treatment and insisted on taking her home on 4th. July, 1942, although they were apprised of the risks. A lumbar



Fig. 38. Case 10 - pyrogram after initial aspiration and instillation of thorotrast. This illustrates clearly the usual size of a temporal abscess, its relation to the middle cranial fossa, and the burr hole used for aspiration.

puncture on 3rd. July showed that the pressure was 150 mm., and the fluid contained 45 mgm. protein and 4 cells.

An X-ray just before her discharge showed the abscess cavity only slightly smaller than after the first aspiration, but its wall was somewhat crenellated suggesting that it was not under tension. This patient has remained well and free from symptoms for the two years which have elapsed since her discharge. She writes that she has led "a perfectly normal life" ever since she left hospital.

Comment : The aspirations in this case were undertaken as preliminary to the eventual extirpation of the abscess. The refusal of further treatment, however, made it necessary to leave this case as one treated simply by aspiration, and that the patient has remained well for two years up to the present can only be taken as an interim recovery. It is possible, of course, that she may never have any further trouble but it is also possible that the infection may flare up again in the future. Such a case should be kept under observation, but the patient's religious persuasion has made that impossible.

Case 11. Streptococcal abscess of left temporal lobe due to chronic mastoiditis. Aspiration of abscess. Bony decompression and drainage of extradural abscess. Permeatal mastoidectomy. Infection of bone flap and subsequent removal. Recovery.

Mr. A.H., aet. 50 (R.I. 25661) admitted 1st September, 1941. He had had intermittent discharge from the left ear since childhood, and in the last 6 months before admission a good deal of pain in and around the left ear. Eight days before admission the pain became much more intense, and his doctor noticed a slight nominal aphasia. In the week before admission the pain persisted and he gradually became drowsy.

On admission he was drowsy, but could be roused to co-operate. There was no demonstrable aphasia. He complained of severe pain in the left side of the skull, but denied any generalised headache. There was a foetid discharge from the left ear and some tenderness on pressure over the mastoid. There was an upper right homonymous field defect, and slight weakness of the right side of the face, but no other neurological abnormalities. There was no papilloedema. The spinal fluid pressure was normal, but the fluid contained 180 mgm. protein and 128 cells, most of which were lymphocytes.

The diagnosis of left temporal abscess was made, but as the intracranial pressure did not seem to be raised it was decided to keep him under observation. The pain persisted, and in the next three days he became a little more drowsy and the aphasia much more pronounced.

On 4th September a burr hole was made above the left ear. There was no extradural pus and the dura looked to be normal, although it was tight. When it was excised the underlying brain began to protrude and there was no subdural pus. A brain needle was introduced into the temporal lobe, and at a depth of 2 cm. the needle was felt to slip

into a cavity from which 5 c.c. of thin, greenish-yellow pus were aspirated. One c.c. of thorotrast was instilled and the X-rays showed a small abscess in the temporal lobe (Fig. 38a). A streptococcus was seen in the direct film of the pus, but cultures were sterile.

He was not relieved by the aspiration. On the following day he was very restless, and the aphasia was so marked that it was impossible to communicate with him. There seemed to be a complete right homonymous hemianopia, and there was a marked right hemiplegia. As the abscess was so small, it was felt that there was little to be gained by a further aspiration, so an osteoplastic flap was reflected over the left temporal region (Fig. 38b). There was some purulent extradural granulation tissue over the base of the mastoid at the lower margin of the flap, but the dura elsewhere was normal. It was not tight, so it was decided to watch the effect of the bony decompression before doing anything else to the abscess. He improved slightly in the next two days, and it was felt that the mastoid infection should be dealt with because of the extradural infection found at the reflection of the bone flap. Mr. R.G. Macbeth did a permeal mastoidectomy on 7th September. His general condition continued to improve: he no longer seemed to be in pain, although the aphasia was still so marked that he could not express himself. The hemiplegia began to recover.

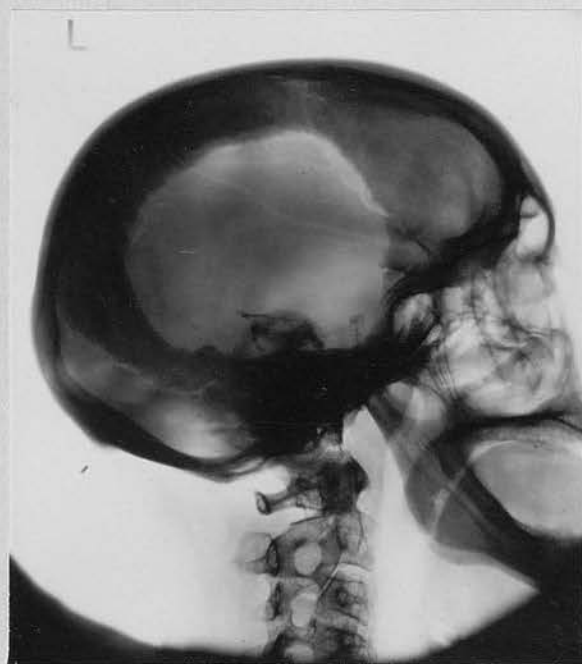
On 11th September, one week after the reflection of the bone flap, the scalp wound was red and inflamed, and on the following day the wound began to discharge pus. An X-ray of the skull at this time showed that the abscess had enlarged and that the bone flap was "riding" slightly (Fig. 38c). His general condition continued to improve, as did the aphasia. The discharge from the wound lessened,

Fig. 38 overleaf. Case 11.

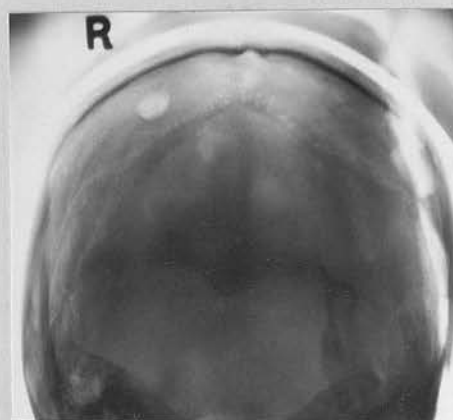


Fig. 38. Case 11 - Pyrogram after initial aspiration, showing the small size of the abscess. Note the site of the burr hole used for the aspiration.

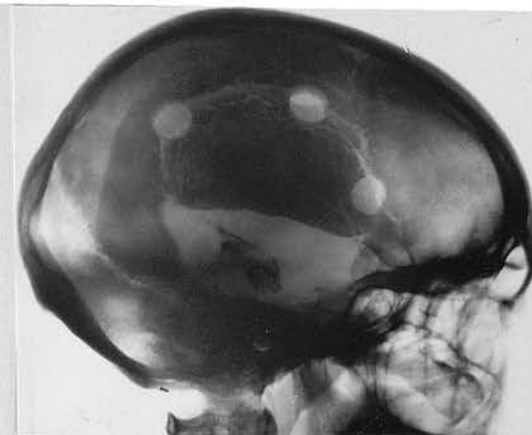
The skiagram at the right shows the bone flap and the increase in the size of the abscess. This plate was taken four days after the initial aspiration (8:ix:42).



Pyrogram 15:xii:42, showing the shrivelling of the abscess, and the angulation of its walls. Note the removal of the bone flap.



Pyrogram 28:ix:42. Note the elevation of the bone flap.



Pyrogram 19:x:42. Note the infection of the bone flap; and that the abscess is diminishing in size.



Pyograms 23:ii:44, eighteen months after the thoro-trast was first instilled. Note that it has shrivelled to a very small angular mass. Cf. Fig. 39.

and we had hopes that the infection would resolve spontaneously, but by 20th October X-rays showed extensive infection (Fig. 38d) of the flap, and it was removed on that day. The abscess meantime had been shrinking, and by 1st November he was free from symptoms and had no neurological abnormalities. The aphasia had cleared up completely, as had the hemianopia and hemiplegia. The spinal fluid pressure was **normal**, and the fluid contained 40 mgm. protein, 8 cells. The scalp wound had healed except for a gap of about 1 cm. in its middle limb which was covered by clean granulations. He asked to be allowed to go home for domestic reasons, and he was discharged on 13th November. He reported on 15th December, free from symptoms, the wound had healed soundly, and an X-ray showed that the abscess had shrivelled up (Fig. 38e). He resumed work on 16th December, and has remained well since then. An X-ray taken 6 months after the aspiration shows that the abscess has shrivelled to a very small size (Figs. 38f and 38g).

Comment: We were misled by the pain in this case, which was probably due to the mastoid infection rather than to increased intracranial pressure due to the small abscess. Once the bone flap was infected it would have been unsafe to open the dura for removal of the abscess. The improvement in his general condition, the disappearance of the neurological abnormalities, made the need for dealing with the abscess less urgent, and by the time the scalp flap was free enough of infection to make an intradural operation safe the thorotrast shadow indicated that the abscess had resolved.

Case 12. Right Cerebellar Abscess (pneumococcus, Type I) due to acute mastoiditis. Suboccipital craniectomy, with repeated aspirations of abscess through unopened dura. Recovery.

M.V., female, aet. 16 (R.I. 7826) was admitted to the Radcliffe Infirmary on 17th April, 1940, when she had an acute otitis media on the right side. On 4th March a myringotomy was done, with relief of pain, but on the following day she had pain in the left ear and a myringotomy was done on that side.

On 8th March there was tenderness over the right mastoid, so a Schwartze operation was done and pus was found throughout the mastoid. The lateral sinus and the dura over the middle fossa appeared to be normal.

From the time of this operation she complained of severe headache, vomited frequently, and was drowsy. A lumbar puncture on 19th March revealed clear, colourless fluid containing 14 cells (lymphocytes) and 80 mgm. protein. Cultures were sterile. By 25th March the tympanic membranes had healed and the mastoid wound looked healthy, but the vomiting persisted, and on this day it was noticed that there was some nystagmus on looking to the right side. The headache altered in character: from being a continuous dull generalised ache, she began to have paroxysms of acute headache, lasting several minutes at a time and occurring once or twice a day, situated at the back of the head, with long intervals of freedom, although she was always aware of some pain in and stiffness of her neck.

By 17th April there had been no improvement. The spinal fluid pressure was 260 mm.; the fluid contained 60 mgm. protein and 10 lymphocytes. There was nystagmus on looking to the right, and

arrangements were made for her transfer to the Radcliffe Infirmary as suffering from a cerebellar abscess.

On admission, she had a good physique, but she looked flushed and ill. The pulse rate was 66/min., and the temperature 99°. She was drowsy and complained of severe headache, but she gave a clear account of her symptoms and co-operated well on examination. She preferred to keep her head bent forward and to the left, and resented any attempt to alter this posture. The mastoid wound was healed, and there was no discharge from the ears.

Her speech was "thick" and slurred, but there was no difficulty in swallowing. There was bilateral papilloedema (2 D.) and no defect in the visual fields. There was slow, coarse nystagmus on looking to the right, and difficulty in maintaining deviation to the right. The nystagmus was more rapid and finer on looking to the left. The ocular movements otherwise were normal. There was no trigeminal or facial paralysis, and hearing was normal in both ears. There was no paralysis of the tongue or palate. There was generalised hypotonia of all limbs, more marked on the right side, and a wild ataxy of the right arm and leg. The tendon reflexes were all less brisk on the right side than on the left, and both plantar responses were flexor.

It was thus clear that there was an expanding lesion in the right cerebellar lobe, and the history and spinal fluid findings left little doubt that it was an abscess.

On 18th April a suboccipital craniectomy was performed, as for the exposure of a cerebellar tumour. The left ventricle was tapped in the early stages of the operation and it was found to be capacious, suggesting obstructive hydrocephalus. When the bone was removed from the

posterior fossa, the dura was very tense and it bulged much more over the right cerebellar lobe than over the left. A small incision was made in it, and a brain needle was passed forward into the right lobe of the cerebellum. At a depth of 1 cm. from the dura the slight resistance of the abscess capsule could be felt. Twenty c.c. of thick yellow pus were aspirated slowly, and 2 c.c. of thorotrast injected into the cavity. The aspiration slackened the tension in the posterior fossa, and after irrigating the wound with proflavine it was closed in the usual manner. Pneumococcus, Type I, was cultured from the pus.

There was a striking improvement following operation. Headache was relieved, the vomiting stopped, and she was much brighter mentally. X-rays showed the abscess cavity in the right cerebellar lobe attached to the posterior aspect of the petrous bone (Fig. 39).

On 22nd April, 4 days after operation, a routine lumbar puncture revealed turbid fluid under a pressure of 220 mm. The fluid contained 80 mgm. protein and 1,500 cells, mostly neutrophils. There were no organisms in direct films or on culture. This burst of meningitis was surprising, in view of the fact that she seemed well in herself, she was afebrile, and had only a slight Kernig's sign in both legs to show for it clinically.

On 25th April, at 2 p.m., she suddenly cried out with severe headache, and her pulse rate shot up from 70 to 140/min. She had marked stiffness of the neck and legs, and it was thought that she had a generalised meningitis. Simultaneous spinal and ventricular punctures were done, the fluid from each source being clear and colourless. The ventricular fluid was normal on analysis (5 mgm.

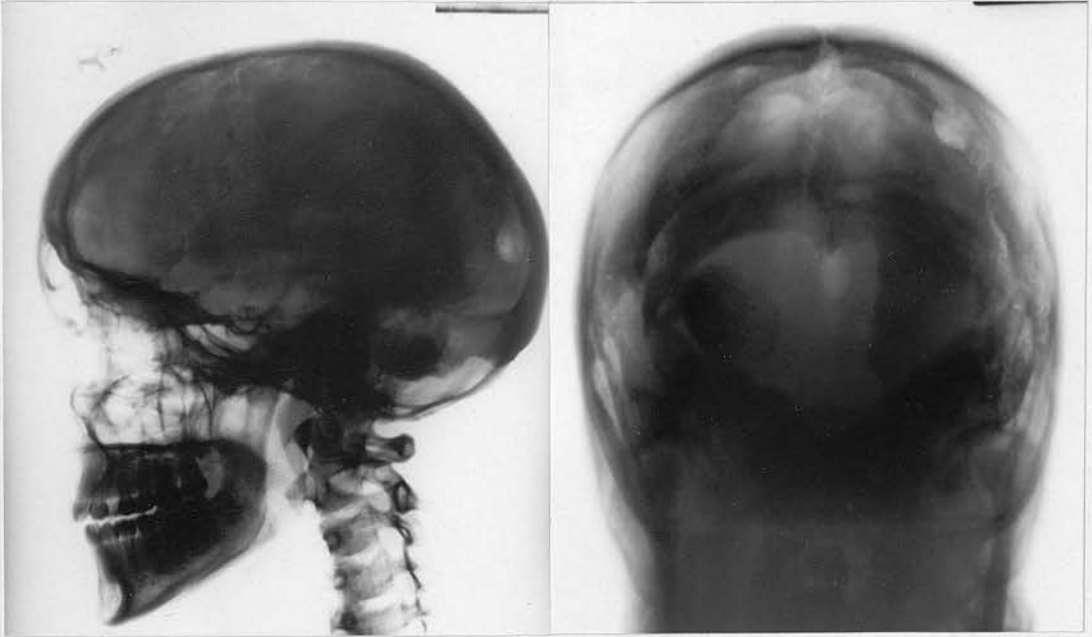
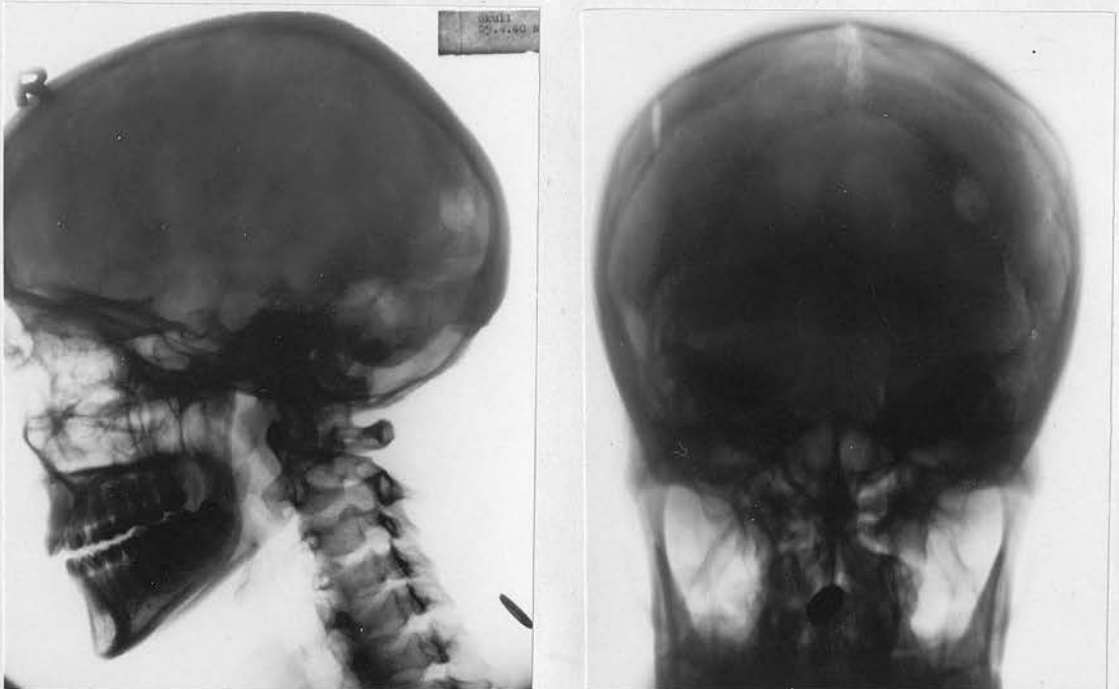


Fig. 39. Case 12 - pyogram 20:iv:40, on the day after the initial aspiration. Note the relation of the abscess cavity to the posterior aspect of the petrous bone.



Pyogram 25:iv:40 - note increase in size of abscess.

Fig. 39 overleaf. Case 12.

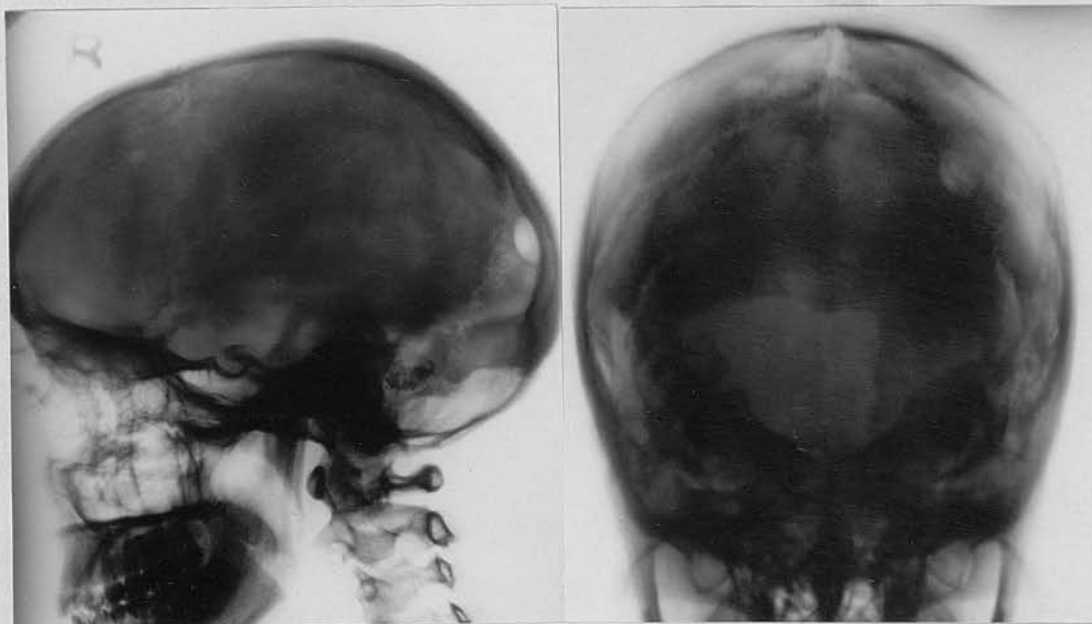
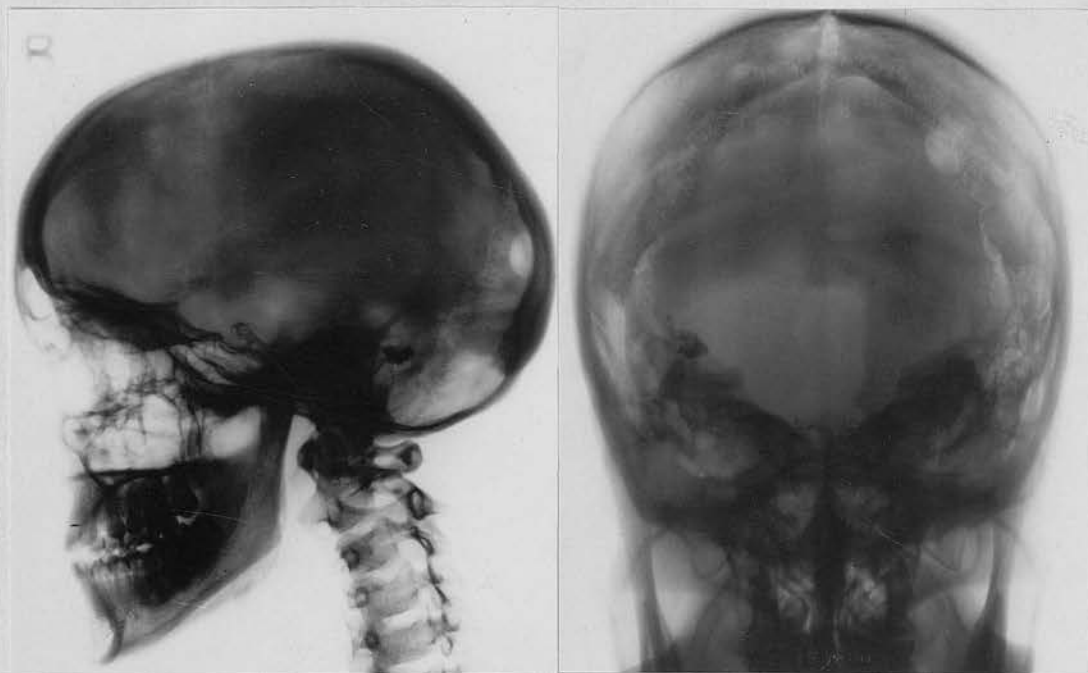
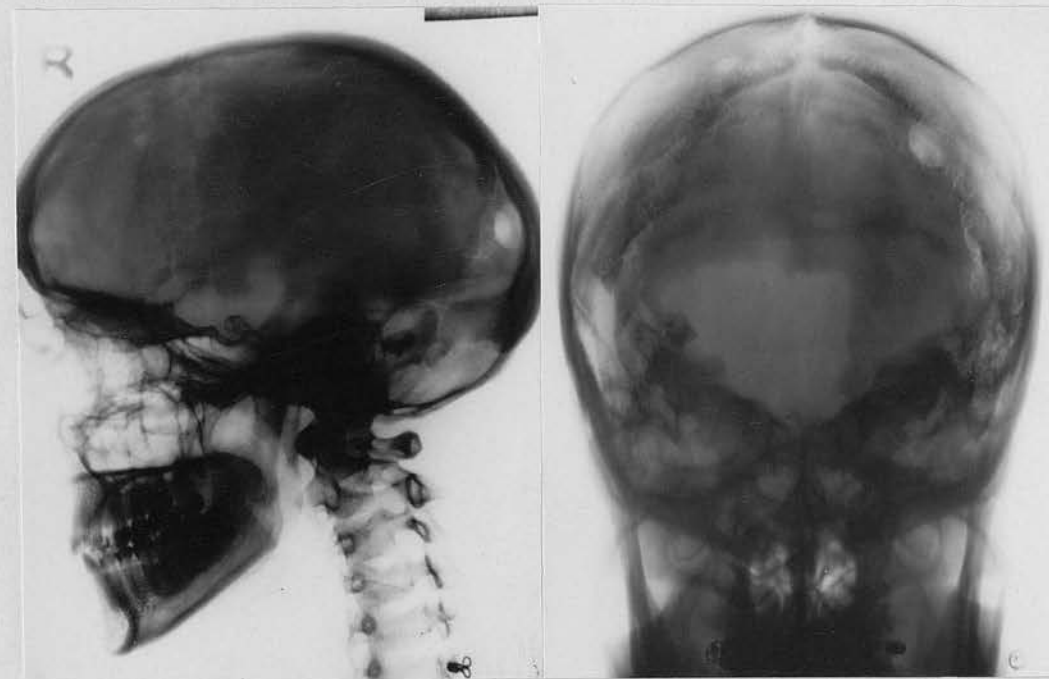


Fig. 39. Case 12. Pyogram 6:v:40.



Pyogram 18:vii:40, showing shrinkage of abscess cavity to a small crenallated mass.



Pyogram 5:vi:40. Note progressive shrinkage of abscess cavity.

protein, 0 cells) and the spinal fluid contained only 20 cells. The pressure in the ventricle was 200 mm., and the removal of 10 c.c. of fluid relieved it to atmospheric level. It was clear that this was a crisis of increased intracranial pressure and that she was not suffering from widespread meningitis.

The headache was relieved by the ventricular tap, but the X-rays (Fig. 39) showed that the abscess had increased in size. Accordingly, on 25th April (one week after the initial aspiration) a sharp needle was inserted through the scalp and muscle flap and another 20 c.c. of pus were aspirated from the abscess.

From this time there was steady improvement. The headache was relieved; the temperature, pulse rate, and respirations settled to normal 48 hours after the aspiration, and there was progressive lessening of the nystagmus and the right-sided cerebellar signs. She started getting up on 6th May, by which time there was no demonstrable ataxy in arm and leg, and the nystagmus was only apparent on maintaining the deviation of the eyes to the extreme right, and the papilloedema had subsided. Further X-rays (Fig. 39) showed a progressive shrinkage of the abscess cavity. The spinal fluid pressure was normal, and although the protein content was normal (35 mgm.) there were still 8 lymphocytes per c.mm.

In the meantime the mastoid wound had broken down (27th April) and there was an intermittent spread to the scalp flap and two sinuses appeared in it. These wounds required frequent dressings, but as she was otherwise free from symptoms she was discharged from hospital on 29th May, 1940, to report for daily dressings as an out-patient. The final cerebrospinal fluid examination on the day before discharge

had shown 35 mgm. protein and 2 lymphocytes.

She remained well and was leading a normal active life until early August, 1940, when she again began to complain of headache, at times severe enough to make her cry, and she vomited on two or three occasions when she had a headache. These symptoms led to her readmission on 20th August, 1940. She looked well, pulse, temperature, and respirations were normal, but she was observed in several bouts of severe headache during the first two days in hospital. The wounds were healed, but the right end of the scar was inflamed and tender to touch. There were no neurological abnormalities; in particular, no papilloedema, no nystagmus, and no ataxy. The thorotrast shadow (Fig. 39) was shrivelled up. Despite these signs, it was thought that there was probably another abscess, or a loculus which had not been tapped previously. The fact that the spinal fluid contained 100 mgm. protein and 5 lymphocytes lent some weight to this assessment.

The scalp flap was reopened on 22nd August, 1940. There was a small sequestrum floating in a loculus of pus in the right limb of the incision (from this, as from all of the wound sepsis in the earlier stages of the illness, staphylococcus aureus was grown in pure culture). As the cerebellar abscess had grown nothing but pneumococci, the wound infection was probably a contamination, as there were other cases of staph. aureus infection in the ward at the time. The dura over the cerebellum was quite slack. An exploring needle inserted in the right cerebellar lobe met only the stony resistance of the thorotrast-encrusted scar, and further attempts revealed only a dilated fourth ventricle from which clear, colourless fluid (140 mgm. protein, 5 lymphocytes) was obtained. There was no pus. The superficial sinuses were

curetted, and the wound was closed.

Again she recovered completely. By 9th September, 1940, she was free from symptoms and signs, the spinal fluid pressure was normal, and the fluid contained 25 mgm. protein and no cells. The scalp wounds took another eight weeks to heal finally, but she has remained well during the $3\frac{1}{2}$ years which have passed since her discharge from hospital.

Case 13. Chronic left cerebellar abscess due to chronic mastoiditis. Decompression.

R.L., male, aet. 32 (R.I. 20237) was admitted to the Radcliffe Infirmary on 27th December, 1941. At the age of 18 he had had an acute illness which was thought to be cerebrospinal fever. He recovered completely and was well until, at the age of 20, he had an acute middle ear and mastoid infection for which a radical mastoidectomy was done. From that time there had been an intermittent discharge from the left ear, although there was never any pain or discomfort in the ear or mastoid area.

About six months after the mastoid operation he began to have attacks of very severe generalised headache and vomiting lasting for two or three days at a time. These attacks were prostrating while they lasted, but as he had long periods of complete freedom with excellent general health they were regarded as migrainous phenomena. Indeed, up until April, 1941, he had these attacks of headache only once or twice a year. At this time he had a more protracted attack which kept him off work for five weeks, and it differed from the others in that while the headache lasted he had diplopia on looking to the left. From that time the headache was more frequent, although not as severe as in the acute attacks. He often woke with a headache in the morning, or it would be brought on by stooping or straining. He vomited occasionally, and his wife noticed that his personality was altering in that he was becoming much more irritable, and he occasionally seemed to be a little confused.

Three weeks before admission he began to be slightly unsteady in walking, and his doctor detected some nystagmus on looking to the left

side. The headache and unsteadiness of gait made it necessary for him to give up his work.

On admission to hospital he was complaining of very severe headache across the brow and at the back of the head. He was quite conscious and rational, and there was no dysarthria or aphasia. He kept his head bent forward and to the right side, and resented any attempt to alter this posture, saying that it made the headache much worse. The optic fundi were normal and there was no defect in the visual fields. There was slow, coarse nystagmus on looking to the left, with marked difficulty of maintaining deviation to this side. The nystagmus on looking to the right was less marked, more rapid, and finer. Facial sensibility was intact, but there was slight weakness of the left side of the face of the lower motor neurone type. He was very deaf in the left ear, and the otological examination by Mr. R.G. Macbeth revealed a slight discharge from the mastoidectomy cavity on the left side which he regarded as being due to a persisting low-grade infection. The cranial nerve functions were otherwise normal, but there was very slight hypotonia, dysdiadokokinesis and ataxy of the left limbs. The reflexes were all normal, and there were no sensory defects. His gait was a little unsteady: he walked on a wide base without any lateral deviation, but he tended to lurch to the left in turning quickly, and he did not swing his left arm as freely as the right one. X-rays of the skull showed no definite abnormality except the mastoidectomy cavity on the left side.

On 30th December a lumbar puncture was done during a bout of very severe headache. The pressure was only 160 mm., and in view of his statement that the headache was less severe if he kept his head bent

forward, the head was gently extended and immediately the pressure rose to 300 mm. and stayed at this level as long as the head was kept in this position. When it was bent forwards the pressure rapidly fell to 160 mm. The fluid was clear and it contained 40 mgm. protein and 3 cells. The removal of 5 c.c. brought about striking relief of the headache.

There was little doubt that this patient was suffering from a chronic left cerebellar abscess, but unusual features were the length of history, the normal fundi, despite a protracted increase in intracranial pressure, and the very slight changes in the spinal fluid (3 cells).

On 3rd January, 1942, a ventriculogram was done, and this showed symmetrical dilatation of the lateral ventricles, dilatation of the third ventricle (Fig. 40), and displacement of the aqueduct and fourth ventricle to the right side. A suboccipital craniectomy was done on the same day under general anaesthesia. The left cerebellar lobe was a little more prominent than the right, but it was interesting to see that the tonsils were only just engaged in the foramen magnum and not pushed through as in cases of slowly-growing cerebellar tumours. Flexion and extension of the head did not alter these relations, as had been expected from the observations made during lumbar puncture.

An exploring cannula was inserted into the left lobe of the cerebellum, and at a depth of 5 cm. the resistance of the abscess wall was met. The blunt needle would not pierce it, and by "feeling" in two or three more places it was inferred that there was a small chronic abscess. It was felt that the decompression might relieve his headache and allow the abscess to expand posteriorly into the decompression

Fig. 40 overleaf. Case 13.

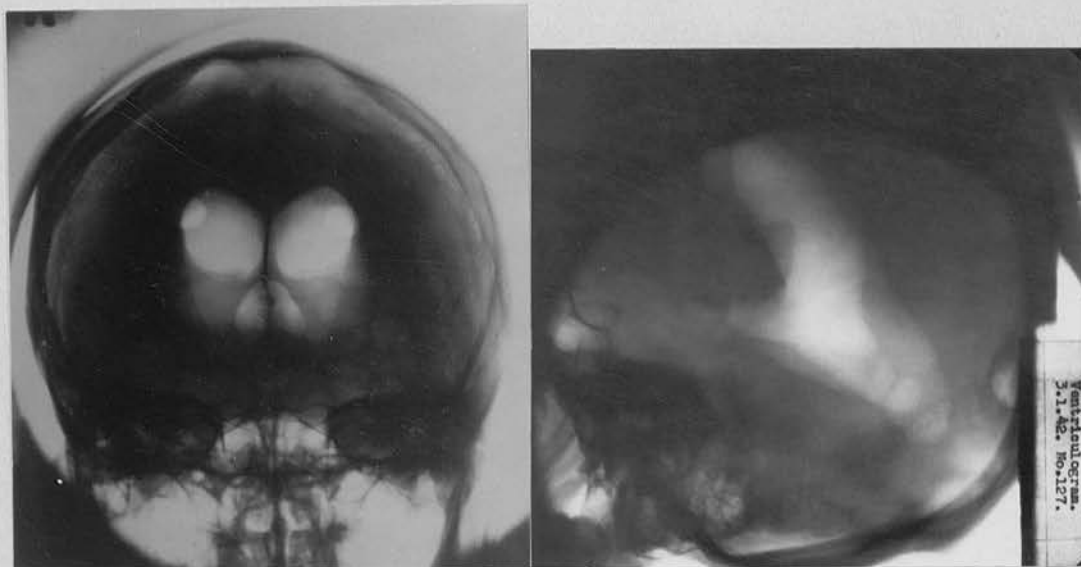


Fig. 40. Case 13 - Ventriculogram, showing dilatation of lateral and third ventricles, forward kinking of aqueduct and failure of filling of fourth ventricle.



Transverse section through cerebellum to show chronic abscess in left cerebellar lobe. Note its position in the upper part of the cerebellum and the extension of the inflammatory process to the anterior surface.



Superior surface of cerebellum. Note the bulging of the upper surface of the left cerebellar lobe, and the groove in the medial part where the upper surface has been herniated upward through the incisura tentorii. This mechanism has as serious potentialities as the more familiar downward herniation of the cerebellar tonsils through the foramen magnum.

opening and thus be more accessible for removal at a later date.

The immediate post-operative result was encouraging. The headache was relieved, there were no new neurological signs, and the wound healed normally. On the 12th day after operation he had some headache, and its recurrence seemed to alarm him. He was a little restless and kept asking for a lumbar puncture, as he remembered the relief afforded on previous occasions. The headache and restlessness got more marked as the day wore on. At 9-30 p.m. a lumbar puncture was done: the pressure was 200 mm., and the decompression was not particularly tense. The removal of 20 c.c. of fluid reduced the pressure to 100 mm., and the decompression became quite slack. Despite this, the headache persisted, and it was thought that the abscess might have compressed the aqueduct, thus causing an increase in intraventricular pressure which might not be manifest in the lumbar theca or the subtentorial decompression. A needle was inserted into the left lateral ventricle and the pressure was normal. At midnight he was given morphia, gr. $\frac{1}{4}$, by hypodermic injection, and this seemed to relieve him. He got to sleep at 2 a.m., and pulse, temperature and respirations at this time were normal. At 5-30 a.m. he suddenly stopped breathing and died within two or three minutes.

At the autopsy, the operative field was normal, and there was no evidence of meningitis. The left lobe of the cerebellum was expanded and its upper surface was impacted in the incisura tentorii (Fig. 40). The herniation in this case had thus been upwards through the tentorial opening rather than downwards through the foramen magnum, as is usually the case. On cutting the cerebellum a thick-walled abscess was found in the anterior part of the superior lobe (Fig. 40). It was adherent

to the dura over the posterior aspect of the petrous bone.

Comment: It was thought that the unusual kind of herniation in this case had some bearing on the character of the headache. Impaction in the incisura tentorii may compress the aqueduct and hinder the passage of cerebrospinal fluid into the fourth ventricle, but it also obliterates the cisterna ambiens and obstructs the passage of fluid upward into the supratentorial compartment of the subarachnoid space. These combined effects may cause a catastrophe within a few minutes by local pressure on the vital centres in the brain stem. Conceivably the morphia favoured such a state of affairs, and its use should be avoided in these cases.

The abscess was not a large one as cerebellar abscesses go: it probably contained 8 c.c. of pus. Its wall was so thick that it was unlikely to have expanded into the decompression, and the proper treatment would have been to aspirate it with a sharp needle. In such a chronic abscess a single aspiration might have sufficed.

Case 14. Left frontal abscess due to acute frontal sinusitis. Aspiration, injection of penicillin, eventual extirpation. Recovery.

Mr. J.W., a prison supervisor aet. 49 (R.I. 20773) was admitted to the Radcliffe Infirmary on 22nd January, 1942. Two weeks before admission he had a severe cold in the head, with a good deal of nasal discharge. About 3 days after the onset of this cold he began to complain of severe pain in the left side of the forehead. This pain was associated with tenderness on pressure, but after five or six days the local pain was gradually replaced by generalised headache, with occasional vomiting. His wife noticed that his manner had become a little strange in the day or two before admission: normally a quiet man, he became talkative and frivolous and at times very confused. There had been some attacks of twitching of the right side of the face, associated with an inability to speak properly for five or ten minutes at a time, but he had never lost consciousness, and in the intervals between these attacks his ability to speak seemed normal. He was right-handed. His temperature had varied between 99° and 100° from the beginning of his illness.

On admission he was conscious, but confused, and there was a moderate expressive aphasia, with a marked tendency to perseveration. He admitted to no headache, although his wife said that just before he came into hospital he had been "in agony" and had asked for poultices to be applied to his head. The temperature was 100°, pulse rate 68/min., respiration 20/min. The general examination revealed marked aortic incompetence, with hypertrophy of the left heart and pulsation of his whole body of a degree to be transmitted to the bed. The blood

pressure was 180/90. The heart lesion was known to have been a sequel of rheumatic fever.

Apart from the mild aphasia and a doubtful extensor plantar response on the right side, there were no neurological abnormalities. The cerebrospinal fluid pressure was 180 mm. c.s.f., the fluid was slightly turbid, containing 224 cells (mostly neutrophil leucocytes) and 50 mgm. protein.

Clinical and radiological examination of the nasal sinuses indicated empyema of the left frontal sinus, and on 23rd January, 1942, Mr. R.G. Macbeth drained a large quantity of pus from this cavity by an incision in the left brow and osteotomy of the anterior wall of the sinus. From the pus a pneumococcus (Type XXI) was grown. This operation seemed to relieve the local pain, but by 25th January, 1942, it was noticed that he was getting more drowsy and that the aphasia was more pronounced. By 27th January there was definite weakness of the right side of the face, and on the following day the weakness had extended to involve the right arm and leg. By 29th January he was very drowsy, incontinent of urine, and had a marked right hemiplegia.

The diagnosis of an abscess in the left frontal lobe was fairly clear. On 29th January his condition admitted no further delay, so a burr hole was made in the left frontal region and 30 c.c. of thick yellow pus were aspirated through a hollow needle. Two c.c. of thorotrast were instilled into the cavity for radiographic delineation of the abscess (Fig. 41a). The effect of this aspiration was to render him more alert immediately, but the aphasia and hemiplegia were unaltered. On 31st January he again became comatose, and a further aspiration had to be done: on this occasion 35 c.c. of pus

Fig. 41 overleaf. Case 14.

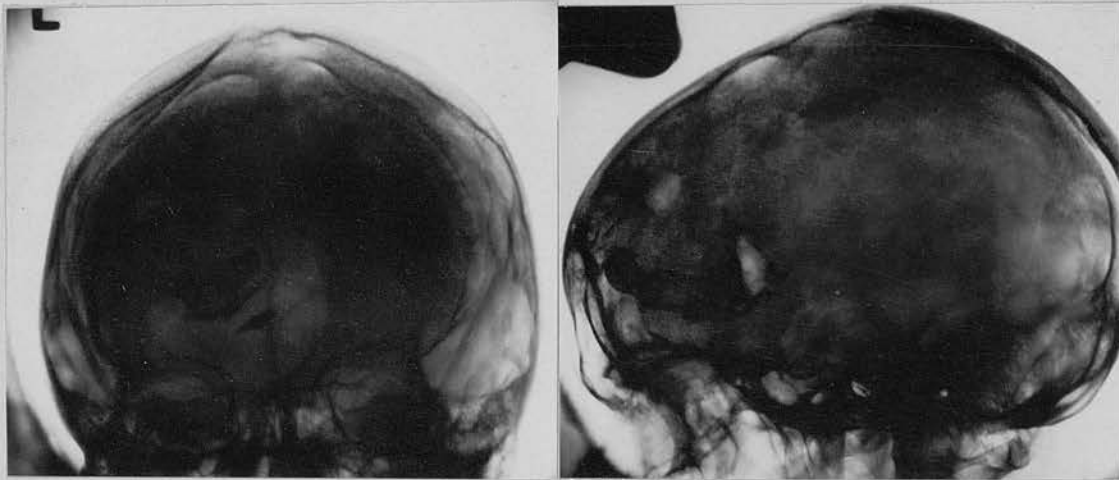
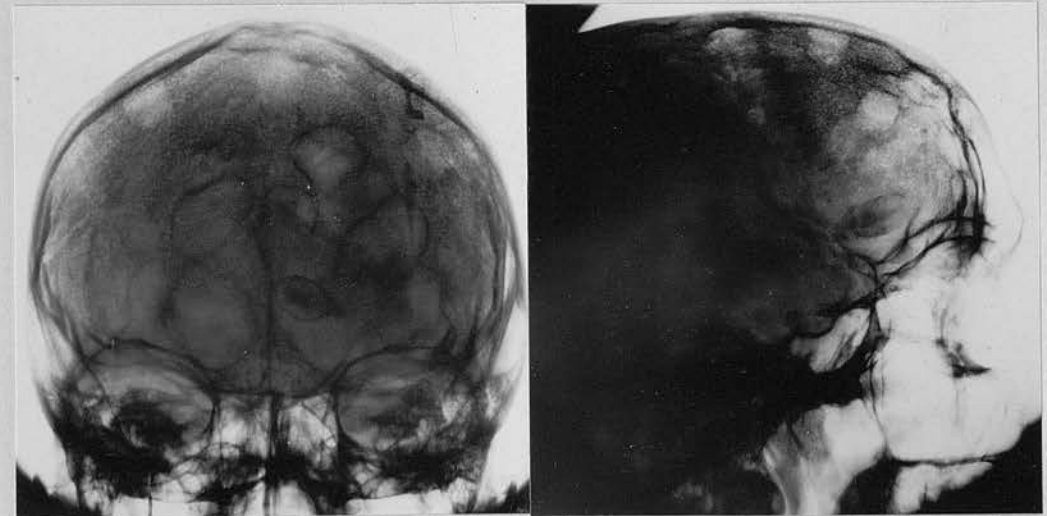
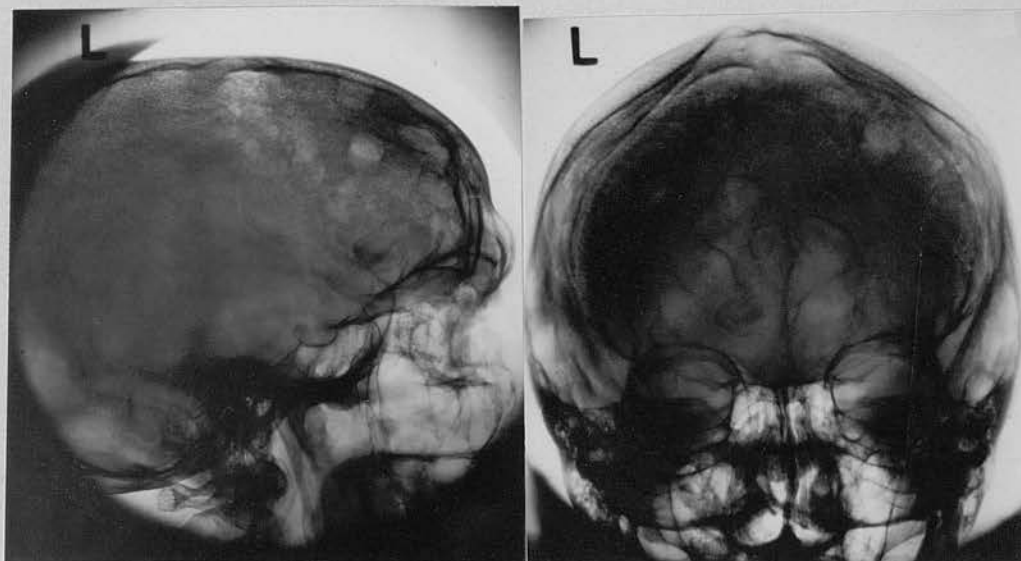


Fig. 41. Case 14 - Pyogram 29:i:41, after initial aspiration and instillation of thorotrast. Note large size of frontal sinuses, which extended almost as high in the vault of the skull as the coronal suture.

Also note the bubble of air in the posterior pole of the abscess cavity.



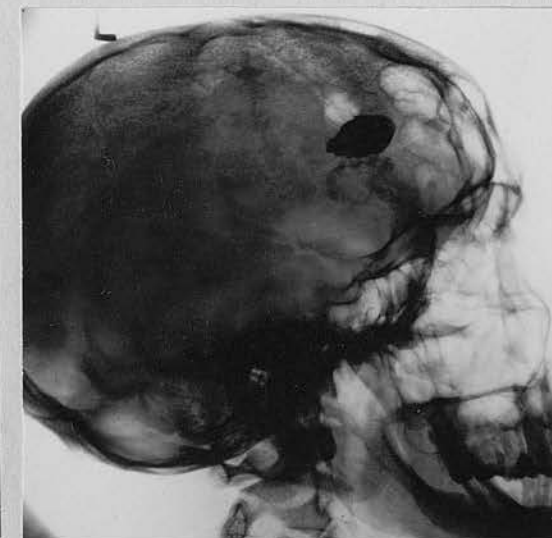
Pyogram 11:ii:42, showing the faint outline of the superficial loculus after an aspiration. The deeper loculus is untouched, but has an appearance suggesting tension in its walls.



Pyogram 23:ii:42, showing that all of the thorotrast has been aspirated from the superficial loculus. The small size and angular appearance of the deep loculus led to the interpretation that the abscess was resolving with repeated aspirations.



Pyogram 27:ii:42. More thorotrast was instilled into the superficial loculus.



Pyogram 14:iii:42. Note decrease in size of superficial loculus.

were withdrawn. He improved for about 24 hours, but on 2nd February was deeply comatose, so another aspiration was done, this time removing 40 c.c. of pus. He improved considerably after this, to the point of being more alert, taking his food, and making some attempt to speak. X-rays in the meantime showed that the abscess cavity (outlined by thorotrast) had a large superficial loculus and a smaller deep loculus (Figs. 41b and 41c). Between 7th and 27th February the abscess was aspirated 7 times at intervals of 2-3 days, on each occasion between 12 and 25 c.c. of pus being withdrawn, and half the volume of penicillin instilled. On the last two occasions the pus was sterile. At this time the lumbar pressure was normal and the cerebrospinal fluid contained 45 mgm. protein and 4 lymphocytes. There was considerable improvement in his general condition in that his speech and mentality were practically normal and he admitted to no symptoms. Repeated radiographic examination suggested that the cavity was diminishing in size (Figs. 41d and 41e).

It was thought that this abscess had been cured by penicillin. The aspirations and injections of penicillin were known to have been concerned with the superficial loculus, but it was thought that the penicillin might have penetrated into the deeper loculus and sterilised it. The patient was allowed to go home on 7th March, 1942. He continued to improve for two days, but on 10th March he was a little drowsy and again began to complain of headache. On 12th March he had a generalised epileptic attack which led to his readmission to hospital.

On examination at this time he was drowsy, confused, and had a mild expressive aphasia. There was a very slight weakness of the right side of the face and of the right limbs. The spinal fluid

pressure was 105 mm., but the fluid contained 200 mgm. protein and 9 cells (7 lymphocytes, 2 polymorphs.). X-rays of the skull showed that the superficial loculus was of about the same size as on discharge, but the deeper loculus was slightly larger. An attempt was made to aspirate both loculi: from the superficial one 8 c.c. of pus were obtained, and this was sterile on culture. From the deep loculus 2 c.c. were obtained, and this contained numerous pneumococci. These aspirations produced no material benefit, so on 16th March a left frontal osteoplastic flap was reflected and the abscess mass dissected out in toto. It was an oval mass, measuring 8.6 x 5 x 4 cm. and on section there were found to be three independent loculi, two of which had been visualised by thorotrast. No organisms were found in the superficial (penicillin-treated) loculus, but there were numerous pneumococci in the walls of the deeper loculi and the pus which they contained.

Convalescence was uneventful. The wound healed normally and the cerebrospinal fluid had returned to normal by 27th March. The right-sided weakness cleared up completely and he was discharged free from symptoms and signs on 8th April, 1942. He returned to his former work in June, 1942, and has remained well since then.

Case 15. Left frontal abscess (*streptococcus haemolyticus*) due to penetrating wound of skull. Repeated aspirations of abscess. Eventual extirpation of abscess. Recovery.

T.A., male aet. 7 (R.I. 12187) was admitted to the Radcliffe Infirmary on 24th November, 1940. For two or three months before admission he had had a succession of "septic spots" on his arms and legs, but these were common among the children of his neighbourhood and no attention was paid to them.

On the evening before admission he was watching his father chop wood when a flying splinter struck him in the left side of the forehead. There was a small cut in the scalp just below the hair-line, but it did not bleed, he was not dazed or unconscious, and nothing was thought of the injury at the time. He went to school as usual on the following day, but when he came home he said that he did not feel well, and he vomited at about 5 p.m. Shortly afterwards he had an epileptic attack which began with twitching of the right side of the face and the right upper limb, but was followed almost immediately by loss of consciousness and generalised convulsions. In this state he was admitted to hospital. The head and eyes were deviated to the right side, and although there were generalised movements of the limbs the right side of the face and the right arm and leg were more affected than the left. A lumbar puncture was done during the fit and the pressure was found to be elevated, probably due to respiratory obstruction. The fluid was clear and colourless. The removal of 10 c.c. of fluid stopped the fit and there was no recurrence. Analysis of the fluid showed 20 mgm. protein and no cells.

On the following day he seemed to be quite well. There were no

neurological abnormalities and the scalp wound (which seemed to be a mere scratch about 2 cm. long just below the hair-line in the left frontal region) had a thin dry crust and looked to be healing normally. An X-ray of the skull, however, showed that there was an underlying punctured fracture of the frontal bone (Fig. 42).

On 28th November, 4 days after admission, the scalp wound began to discharge a little pus, and this continued for seven days, when the wound finally healed. Although he was free from symptoms, the fact of superficial sepsis in relation to a penetrating wound made it necessary to keep him under observation.

On 9th December, he vomited several times, and he began to complain of occasional frontal headache for the first time. During the next week he became listless and apathetic, even when he did not have a headache.

On 16th December he was slightly drowsy, but could be roused easily. Temperature, pulse, and respirations were normal, as they had been throughout, except for occasional rises of temperature to 99°. Both optic discs were flushed, although there was no measurable swelling. He had no aphasia and there was no weakness of his face, trunk, or limbs. The reflexes were all normal. The spinal fluid pressure was 120 mm., but the fluid contained 160 mgm. protein and 8 cells (5 lymphocytes). Despite the absence of neurological abnormalities, it was felt that the drowsiness and the cerebrospinal fluid pointed to an abscess of the brain. A linear incision was made just behind the scar of the scalp wound. On stripping the pericranium from around the fracture, a small granulomatous polyp was seen projecting up through the fracture. A circle of bone about 3 cm. in

Fig.42 overleaf . Case 15.

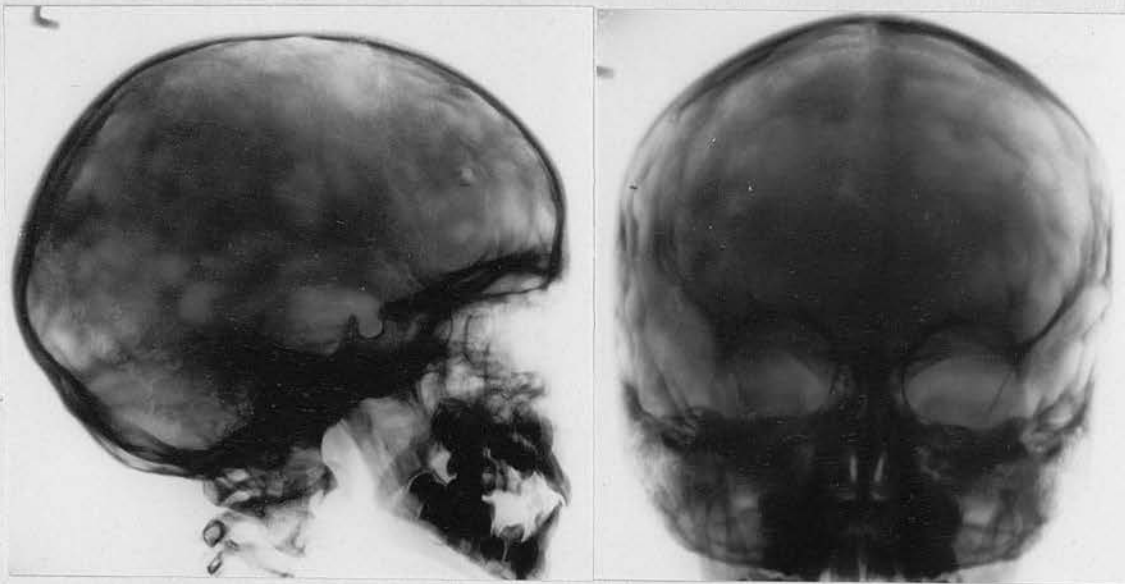
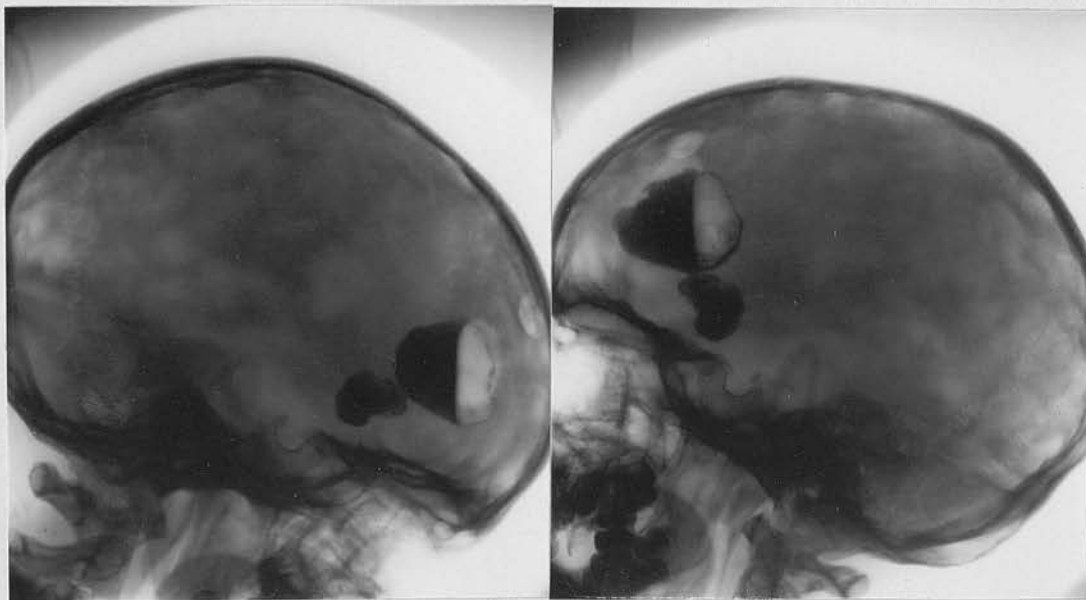
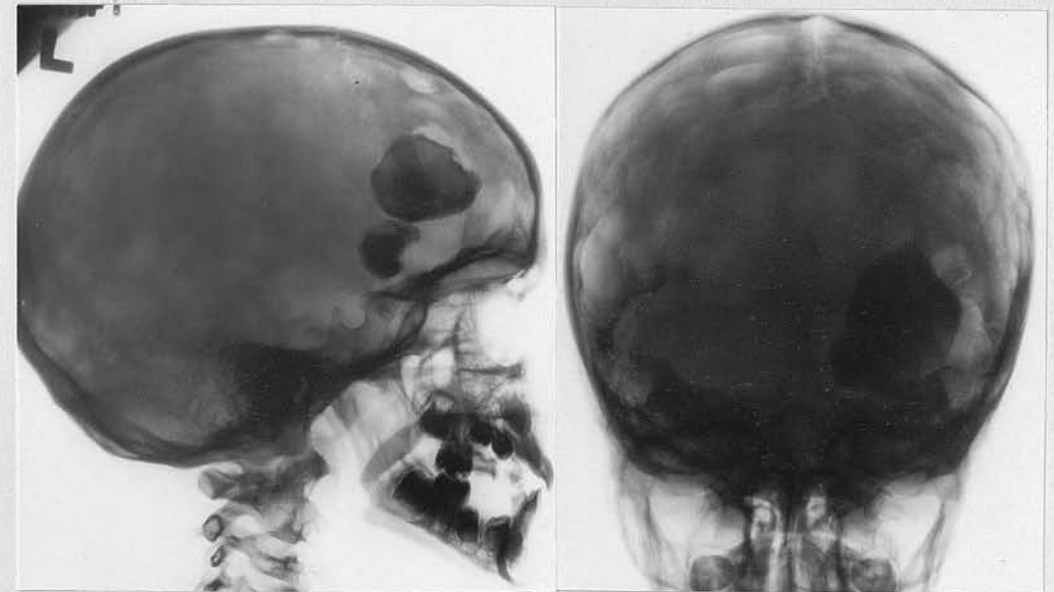


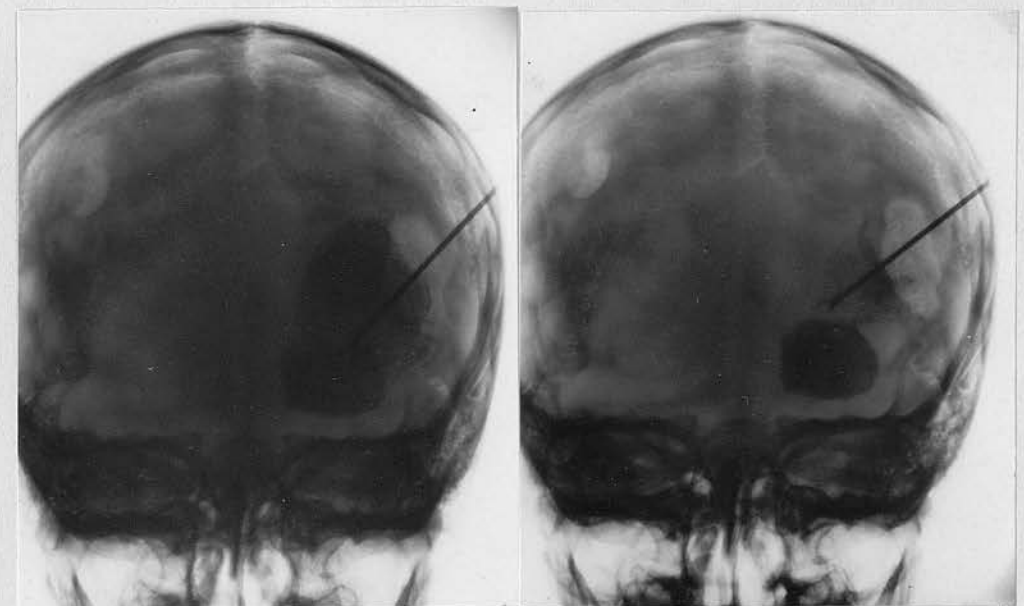
Fig. 42. Case 15 - skiagrams showing small penetrating wound of left frontal bone, with indriven fragments.



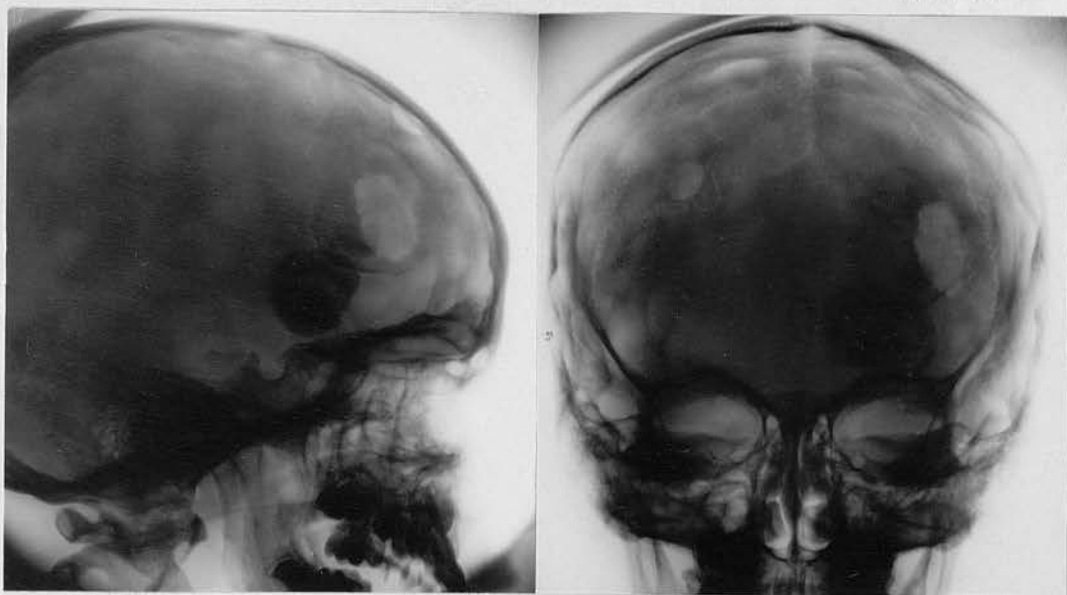
Lateral projections taken in face-up and face-down positions to show bubble of air at anterior and posterior poles of abscess. 17:xII:40.



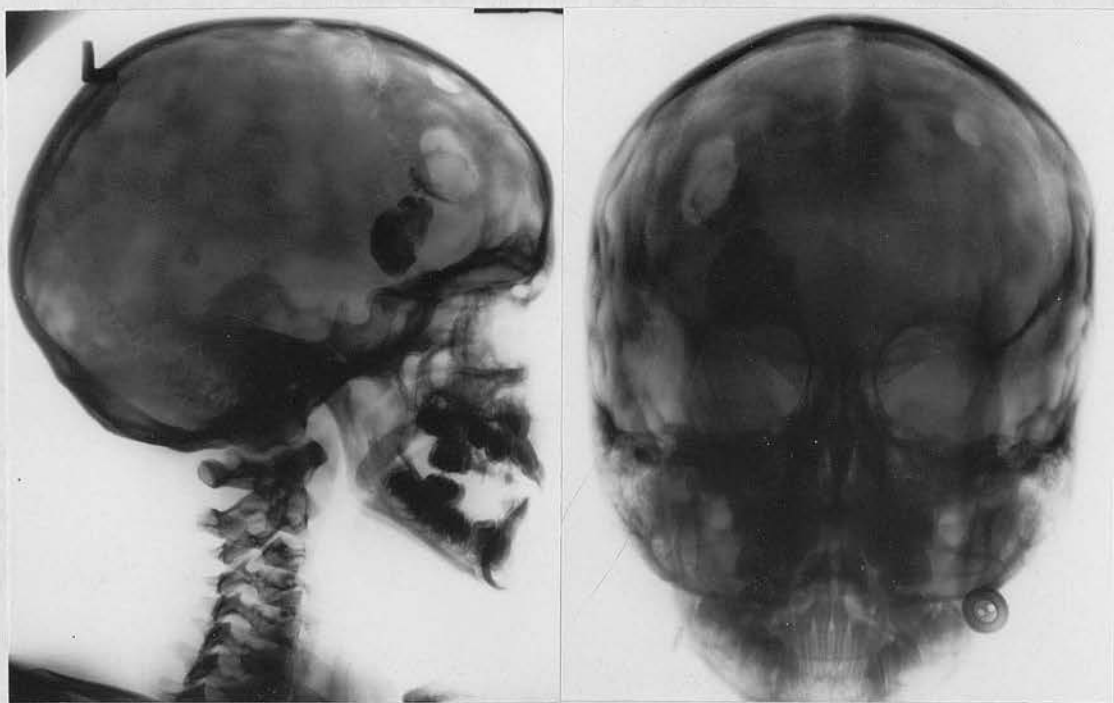
Pyograms 17:xii:40 showing bilocular abscess. These views were taken before the ones at the lower left : in the interval the abscess was aspirated, and the release of tension in the abscess allowed some air to be sucked in, as seen at the lower left.



Pyogram 19:xii:40, before and after aspiration. It will be seen that the needle is only in the superficial loculus.



Pyogram 27:xii:40 - note that the superficial loculus is becoming rather angulated, suggesting that there is no great tension within its walls, while the deeper loculus is increasing in size.



Pyogram 17:i:41. Note shrinkage of superficial loculus, and to a less extent of the deeper loculus. Despite these appearances, there was progressive papilloedema (see p. 221).

diameter was removed around the fracture, and the dura looked healthy except where the granulation tissue was attached to it. A nick was made in the dura behind this attachment, and at a depth of 2 cm. a brain needle entered an abscess cavity from which 15 c.c. of pus were aspirated and 2 c.c. of thorotrast instilled. A haemolytic streptococcus was cultured from the pus. The wound was sutured without drainage after irrigation of the field with peroxide and proflavine. X-rays showed a bilocular abscess in the left frontal lobe (Fig. 42). Headache was relieved and he became more alert, but he continued to vomit, and on 19th December there was slight weakness of the right arm and leg. X-rays showed that the abscess was larger (Fig. 42) and the spinal fluid pressure was over 300 mm. A sharp needle was inserted through the scalp into the cavity and another 15 c.c. of pus were aspirated. Subsequent X-rays (Fig. 42) showed that the larger superficial loculus had been evacuated, but the deeper one had not altered.

Again there was improvement for 2-3 days, but the headache and drowsiness recurred, so on 1st January, 1941, another 12 c.c. of pus were aspirated. From this time there was steady improvement: the headache and vomiting ceased, he became quite bright and active, and the only definite neurological abnormality was a persisting extensor plantar response on the right side. The spinal fluid pressure, however, was always about 240 mm., and the fluid contained 40-60 mgm. protein and 6-8 lymphocytes. The persistence of these abnormalities suggested that the abscess would require further treatment.

On 24th January, 1941, eight weeks after the initial injury, a left frontal flap was reflected. When the bone flap was elevated,

there was a hernia at the site of the initial dural perforation, measuring about 2.5 cm. in diameter by 1.5 cm. in height. The dura was very tight (the operation was done under general anaesthesia) and the right ventricle could not be located for tapping. It was known from the pyograms that the abscess mass was immediately beneath the hernia, so the dura was cut in a circle 5 cm. in diameter around the base of the hernia. The leptomeninges and cortex were cut in a concentric circle 4 cm. in diameter, and the intracranial pressure began to force the abscess mass out through the dural opening. The wall of the abscess was tough enough to make the dissection from the white matter fairly easy, and the whole mass was removed without rupture (Fig. 43). The brain became more slack, and after haemostasis in the cavity the dural defect was repaired with a graft of pericranium, and the wound closed in the usual manner.

The specimen was a pyramidal mass 4.5 cm. x 4 cm. (in cross-section at the base) x 6 cm. high. The base was formed by the extradural hernia surrounded by a cuff of dura and subjacent cortex, immediately deep to which was a triradiate abscess with stout fibrous walls up to 0.35 cm. thick. Cultures of the pus in the abscess were sterile, but all specimens from previous aspirations had grown B-haemolytic streptococcus.

Convalescence was uneventful. The wound healed normally. For three days after operation there was slight weakness of the right side of the face and of the right upper limb, but when he started getting up 7 days after operation and when he was discharged on 6th February, 1941, there were no neurological abnormalities. He returned to school and resumed his place in his own class. There has been no recurrence of the epilepsy in the three years which have elapsed since operation.

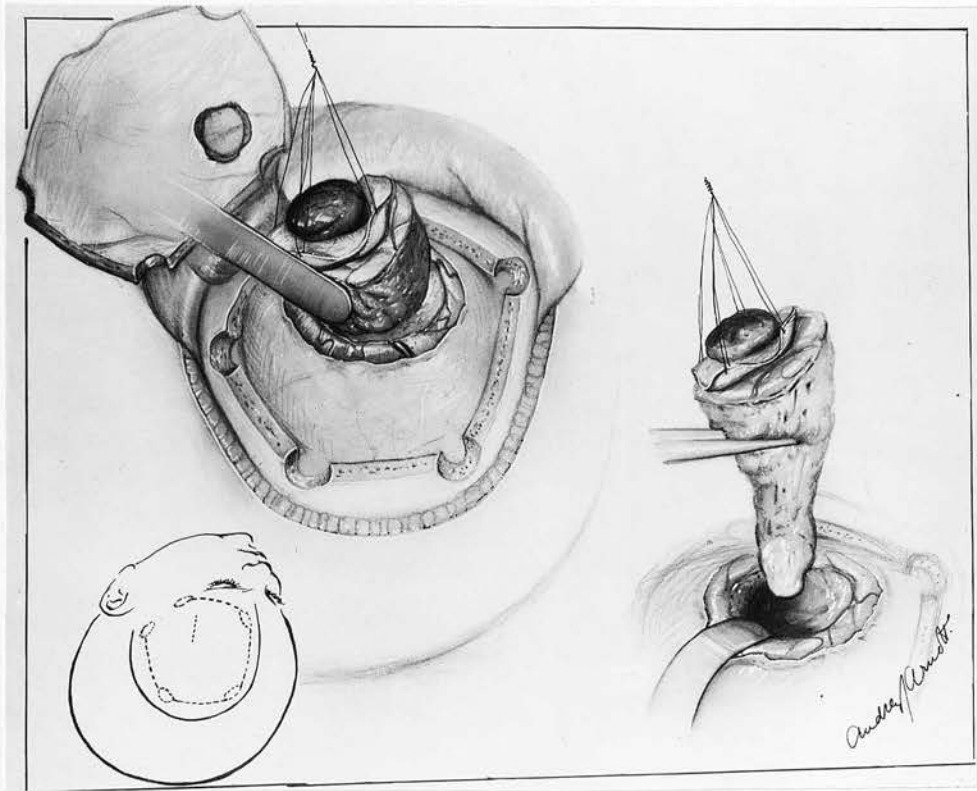


Fig. 43. Case 15 - operation sketch. Note the small hernia cerebri which occurred through the dural incision used for aspiration of the abscess.

Case 16. Right frontal abscess (*staphylococcus aureus*) due to compound fracture of the skull. Aspiration. Extirpation of large multilocular abscess. Recovery.

A.G., female aet. 6 (R.I. 25654) was admitted to the Radcliffe Infirmary on 1st September, 1942, when she fell from a bridge 27 feet high and struck her head in the right frontal region. She was unconscious for about five minutes, and was taken to a hospital where it was found that she had a compound depressed fracture of the vault of the skull, but no other serious injuries. An operation was performed shortly after admission, but no details of the findings or the subsequent progress of the case were obtainable. The child's parents said that the wound healed satisfactorily and that she seemed quite well until two weeks after the injury when some weakness of the left arm and leg was noticed. This weakness became more marked as time went on, and a swelling appeared at the site of the operation. She complained of headache and frequently vomited, but there were periods of two or three days at a time when she seemed to be fairly well except for the progressive paralysis of the left arm and leg. Despite these symptoms she was sent home on 30th August, 1942, and her own doctor arranged for her transfer to the Radcliffe Infirmary.

On admission she was alert and intelligent and cooperated well during the examination. Temperature, pulse, and respirations were normal. There was a cruciate scar in the right frontal region (Fig. 44) in one angle of which there was a tense rounded swelling 4 cm. in diameter projecting about 3 mm. above the level of the scalp. The swelling pulsated and had the characters of a cerebral hernia. There was no papilloedema, and the visual fields were normal.



Fig. 44. Case 16 - pre-operative photograph showing hernia cerebri and the cruciate scar of the original injury.

The child had a complete left hemiplegia, the left limbs were flaccid, the tendon reflexes were exaggerated, and the left plantar response was extensor. The sensory examination revealed no loss of appreciation of light touch and painful stimuli, but postural and stereognostic sensibility were defective in the left arm and leg. X-rays of the skull showed an operative defect in the right fronto-parietal region, with fissures extending from it, one of them back across the vault of the skull into the foramen magnum. There was one indriven fragment at the postero-inferior margin of the defect. The spinal fluid pressure was 300 mm., and the fluid contained 48 mgm. protein and 1 cell. On 12th September she was a little drowsy, complaining of severe headache, and she vomited several times. The hernia was so tense that it no longer pulsated. The spinal fluid on this day contained 65 mgm. protein and 16 cells (60% neutrophils). The postero-superior end of the cruciate incision was opened over intact skull and a burr hole made. A blunt needle was directed towards the base of the hernia and the tough wall of the abscess was encountered. It was so tough that the needle could not pierce it, so a sharp needle was substituted and 15 c.c. of pus were aspirated and 1 c.c. of thorotrast instilled. This slackened the tension of the hernia, eased the headache and vomiting, but the effect was temporary. From the feeling with the blunt needle it seemed that the abscess had a sufficiently thick wall to allow it to be extirpated. On 15th September a horse-shoe flap was reflected with the supraorbital and superficial temporal arteries included in its base, as the pre-existing scar had probably vitiated the blood supply in the vicinity. The scalp was lightly adherent to the leptomeninges over the hernia, and

the dura was absent. This operative defect was roughly circular and about 4 cm. in diameter. The bony opening was enlarged by 1 cm. all round to expose a margin of normal dura. The cortex at the base of the hernia was incised and the wall of the abscess was found to be less than 0.5 cm. from the surface. It was clearly a very large abscess in an eloquent site, and to facilitate dissection another 30 c.c. of pus were aspirated. This partially collapsed the cavity and rendered the subsequent dissection much more convenient.

The abscess was a trilocular mass measuring 10 cm. x 8 cm. x 4 cm. The superficial locus had the thickest wall and it was the most superficial one projecting into the hernia. Deep to it was the largest locus (aspirated during operation), and the third locus seemed to be a bud from this one. The deep surface of the mass was adherent to the meninges along the sphenoidal ridge, and the middle cerebral artery was displaced downwards into the temporal fossa. The dissection from these structures was tedious, and the smallest and deepest locus was ruptured at the end of the dissection. The pus was quickly taken up in the sucker and the field irrigated with pro-flavine solution. When the mass was lifted out it weighed 110 grams. In the large cavity in the postero-inferior part of the frontal lobe the whole course of the middle cerebral artery could be seen, as the Sylvian fissure and insula had been unroofed as far posteriorly as the lower end of the central sulcus. In view of the contamination of the cavity it was thought ~~inadvisable~~ to insert a fascial graft in the dural defect, and the scalp wound was closed in the usual manner.

Convalescence on the whole was uneventful. The wound healed normally, but on the sixth day after operation a pin-point opening

appeared in the old cruciate incision, and this allowed the escape of small quantities of clear cerebrospinal fluid (sterile on culture) for 9 days, after which the wound healed soundly and remained so. The decompression area was indrawn (Fig. 45), but being behind the hair-line it was not unsightly.

The hemiplegia remained complete for ten days. Power then began to return in the left side of the face and left lower limb. Five weeks after operation she could walk unaided; and she was discharged on 5th November, 1942. Three months later the improvement had continued, but there was still marked weakness of the left side of the face, the left upper limb showed only feeble voluntary movements at the shoulder, elbow and fingers, but there was not enough power to render the limb of any use to her. The lower limb had become stronger, but she walked with a marked spastic hemiplegic gait. The sensory abnormalities noted before operation persisted.

Fig. 45 overleaf. Case 16.



Fig..45. Case 16 - post-operative photographs showing
relation of scalp flap to scar of original injury,
and the depression at the site of the craniectomy.
See page 48 and Fig. 10.

Fig. 46 overleaf. Case 17.

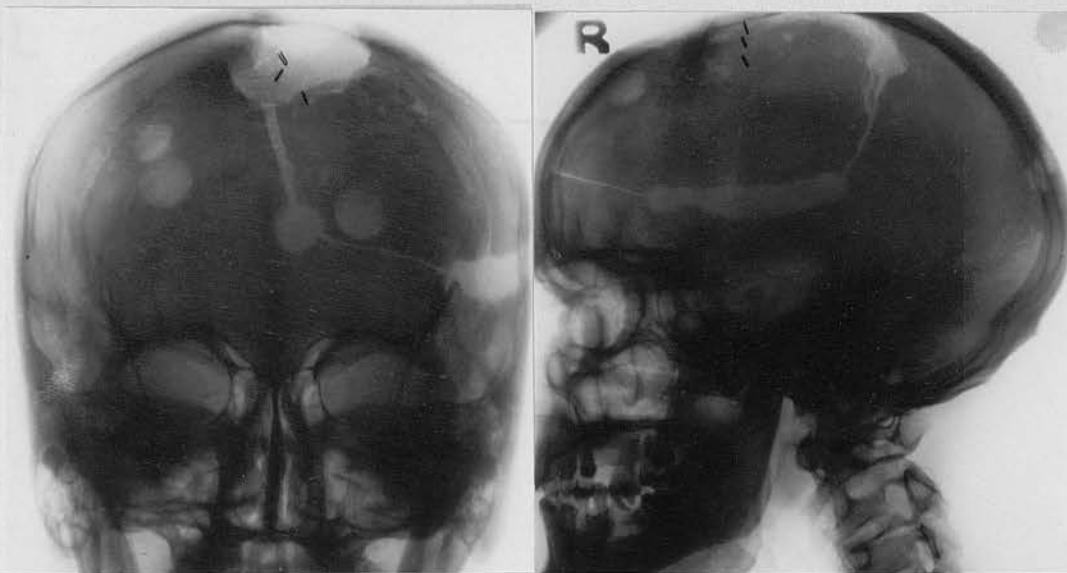
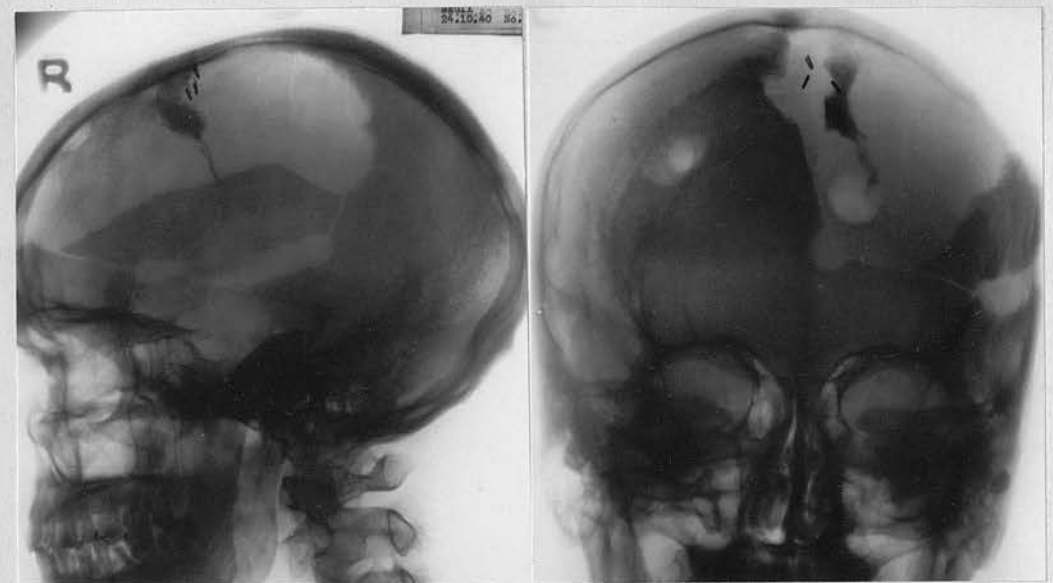
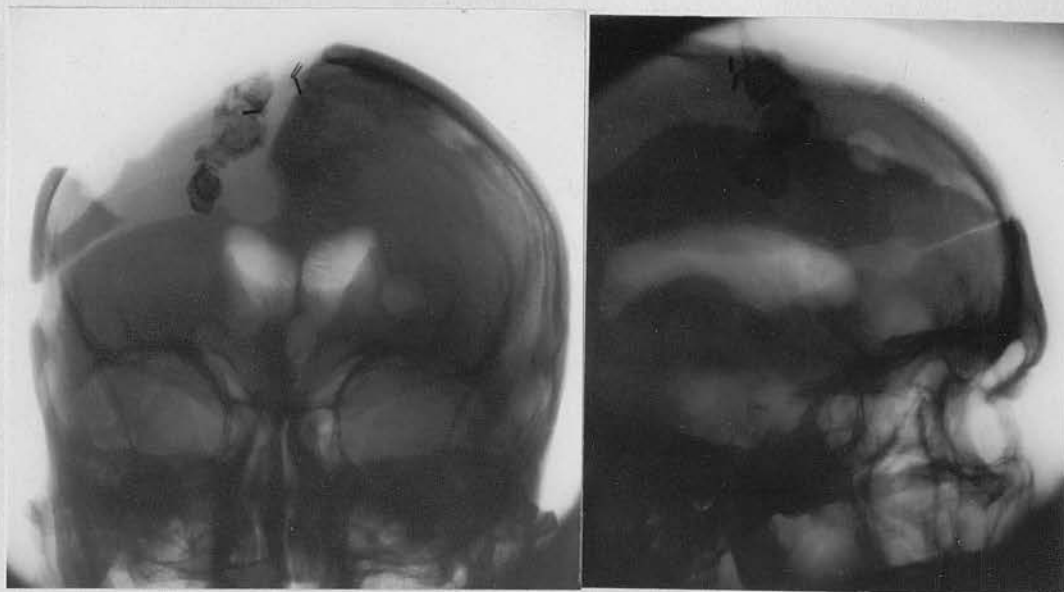


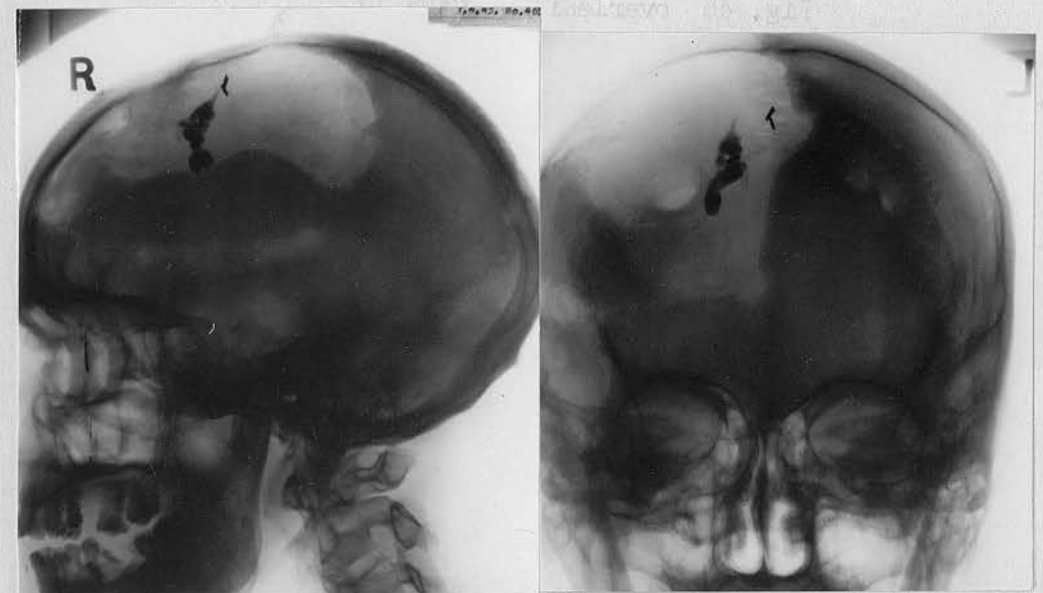
Fig. 46. Case 17 - Skiagraphs 4:x:40, showing osteitis of upper medial part of bone flap, and slight elevation of the flap. Part of the medial margin of the bone flap was excised at the first operation because it was invaded by tumour.



Pyogram 24:x:40, showing thorotrast in the the abscess cavity, and the extent of the sacrifice of the bone flap.



Ventriculogram 20:xi:40, showing slight dilatation of right ventricle and absence of deformity or displacement. This appearance indicated that the abscess had resolved.



Pyogram 7:iv:43, showing that the thorotrast shadow has shrivelled as the result of gliotic contraction of the wall of the abscess. Note that it has retained the outline seen in the plates to the left, but on a reduced scale.

began to protrude as a fungus. There was an occasional rise of temperature up to 100°. A week after the bone flap was removed he said that he felt too ill to get up, but there were no neurological abnormalities and no signs of meningitis. The spinal fluid pressure was 260 mm., and the fluid was slightly opalescent. It contained 864 cells (74% neutrophils) and 100 mgm. protein. By 21st October there was a little stiffness of the neck and slight weakness of the left lower limb. Two days later there was a slight but definite weakness of the left arm and leg, with exaggeration of the tendon reflexes, and the left plantar response was extensor. The neck stiffness had cleared up, however, he felt better generally, and the spinal fluid was clear, although it still contained 90 mgm. protein and 104 cells (82% lymphocytes). The fungus was tense, but its surface was clean.

It seemed likely that there was an abscess in the cavity from which the tumour had been removed. On 24th October a small incision was made in the scalp adjacent to the fungus and a brain needle was directed towards the cavity. At a depth of 4 cm. from the scalp surface there was a slight resistance of the abscess wall. When this was penetrated 12 c.c. of thin yellow pus were aspirated (staph. aureus present in direct films and was grown on culture). Thorotrast, 1.5 c.c., was injected into the cavity (Fig. 46).

The effect of the aspiration was to slacken the tension of the fungus so that its surface became wrinkled. The left hemiplegia had disappeared by the end of a fortnight, but the spinal fluid still contained 14 cells and 65 mgm. protein, although the pressure was normal. By 16th November, the protein content had fallen to 50 mgm. and there

were no cells. This was nearly normal, and, taken together with the improvement in his general condition, the disappearance of the neurological signs, and the fact that the X-rays showed no increase in the size of the thorotrast shadow, it was felt that the abscess might be resolving with the single aspiration. To make certain that there was no residual collection, a ventriculogram was done on 20th November, (Fig. 46) and this showed a slight ventricular dilatation (doubtless the sequel of the mild meningitis) with no deformity, and these appearances were taken as excluding a recollection in the abscess cavity.

The surface of the fungus meantime had epithelialised and it remained slightly indrawn. He was discharged on 29th November, 1940, free from symptoms and with the wound healed. He has remained well since then, and has been working regularly as a general labourer. X-rays taken $2\frac{1}{2}$ years later show the thorotrast in a shrivelled mass (Fig. 46). It will be seen, too, that the position of the shadow has altered slightly, doubtless the result of cicatricial contraction around the mass.

Case 18. Abscess (*staphylococcus aureus*) of right frontal lobe extending into right orbit, due to infected scalp wound. Extirpation of abscess. Recovery.

D.S., male aet. 6 (R.I. 283) was admitted to the Radcliffe Infirmary on 18th January, 1939. He had been a healthy child until 31st October, 1938, when one of his playmates hit him over the right eye with a stone. There was a small laceration at the outer end of the eyebrow which was not sutured or seen by a doctor, as it seemed a trivial injury. The wound seemed to be healing normally, but on 5th November he complained of a good deal of pain over the eye, and the lids became swollen. On 7th November he complained of severe pain and vomited several times. Later on this day he had a generalised epileptic attack lasting for half an hour. He was taken to the Nottingham General Hospital, where a lumbar puncture was done: the pressure was normal and the fluid was normal on analysis. On 12th November, because of an increasing swelling over the right eye, an incision was made in the line of the brow and a small quantity of pus evacuated. *Staphylococcus aureus* was cultured from the pus. The discharge lasted only two or three days, the wound healed, and he became free from symptoms. From that time, however, his mother thought that the right eye was a little more prominent than the left and was placed at a lower level in the orbit.

He remained well for 5 weeks. At about the middle of December, 1938, he began to have paroxysms of very severe headache, lasting for about 30 seconds, coming on and passing off quite suddenly, with freedom from any pain in the intervals. At first these headaches occurred only once or twice a day, but they became more frequent and severe and led to his

transfer to the Radcliffe Infirmary.

On admission he looked healthy and said that he had had no headache for two or three days. He was very bright and alert, and during the six days he was under observation he played about the ward like a normal healthy child. At no time did he complain of or seem to have any headache.

The right eyeball was displaced forward and downwards (Fig. 47). In the medial half of the upper lid there was a hard, pulsating swelling which was not tender, and there were no superficial signs of inflammation. The pulsation was transmitted to the right eyeball. On auscultation a loud rhythmical bruit could be heard over the orbital swelling, and it was transmitted widely over the skull, being loudest over the right mastoid region. This bruit could be obliterated by compression of the right common carotid artery in the neck.

Both optic discs were grossly swollen (5 D.) and there were many haemorrhages in each fundus. The visual fields were normal, except for enlargement of the blind spots. All of the movements of the right eyeball were slightly limited, and in particular upward movement seemed to be more affected than any of the others. Despite the obvious asymmetry of the visual axes, he admitted to no diplopia. In this connection, the acuity in the left eye was 6/15 and that in the right 6/60.

There was a very slight left facial weakness and the left plantar response was occasionally extensor, but there were no other neurological abnormalities.

X-rays of the skull showed an area of erosion in the roof of the right orbit and early separation of the sutures due to increased intra-

cranial pressure. Lumbar puncture on 21st January, 1939, recovered clear fluid under a pressure of 600 mm. The fluid contained 40 mgm. protein and 4 cells. A blood count revealed a leucocytosis of 13,600.

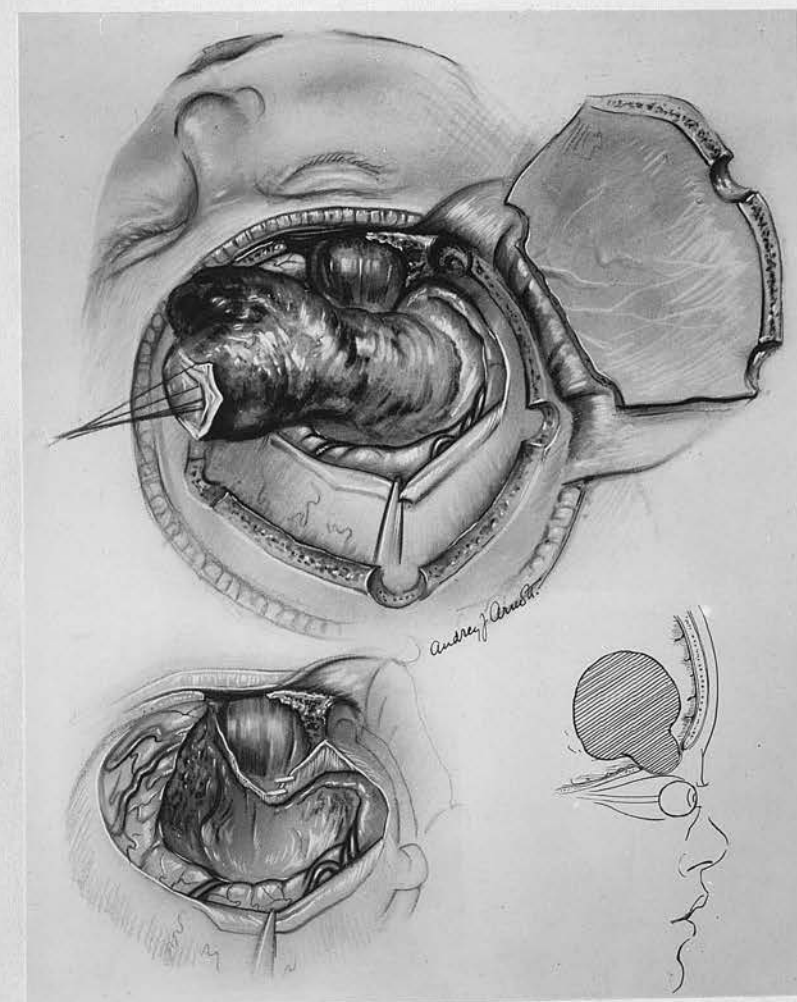
On 24th January, 1939, a right frontal osteoplastic flap was reflected. There was no evidence of sepsis in the scalp or bone. The dura was very tense, and an attempt to lower the pressure by tapping the left ventricle was unsuccessful. Accordingly, a sharp needle was inserted through the dura over the right frontal bone and at a depth of less than 1 cm. the resistance of the abscess wall was met and pierced. Thirty c.c. of pus were aspirated (*staph. aureus* on culture) and this slackened the dura sufficiently to permit it to be opened easily. The convolutions of the anterior part of the frontal lobe were broad and pale, and they were adherent to the dura at the antero-inferior margin of the frontal lobe. A circle of cortex 3 cm. in diameter was removed and this revealed the wall of the abscess. The dissection was carried down to the erosion in the orbital plate of the frontal bone where it was possible to free the wall of the abscess from the dura and to deliver the loculus which had herniated into the orbit (Fig. 47). There was another small loculus extending into the lateral part of the frontal lobe; this was adherent to the main mass, but its walls were thinner and it ruptured during the dissection. The pus was quickly taken up in the sucker with a minimum contamination of the field. The abscess mass measured 8 cm. in its antero-posterior axis by 5 cm. from side to side. The brain was very slack at the end of the operation. A gutta percha drain was inserted into the large cavity and led out through a stab wound behind the posterior limb of the incision.

His convalescence was uneventful. The wound healed normally, but

Fig. 47 overleaf. Case 18.



Fig. 47. Case 18 - X-rays of the skull showing early separation of sutures and convolutional atrophy of the vault. The skiagram at the right shows an area of erosion of the roof of the right orbit : it was through this erosion that one loculus of the abscess protruded into the orbit and caused unilateral proptosis , as indicated in the operation sketch at the right. The defect in the roof of the orbit can be seen in the lower left diagram of the sketch, after the abscess mass had been removed.



three weeks after operation a subcutaneous collection of pus presented in the right brow and required an incision. There was some purulent discharge for several days, and indeed the sinus did not heal finally until October, 1939, after the discharge of a small spicule of bone. He started getting up on the 10th day after operation, by which time the spinal fluid pressure was normal and the fluid contained 50 mgm. protein and 3 cells. The proptosis of the right eye disappeared, but it remained at a lower level in the orbit than its fellow. He never had any diplopia, however. The bruit was still audible in April, 1940, but could not be heard in October, 1940. There have been no further epileptic attacks.

Case 19. Right temporal abscess (*staphylococcus aureus*) due to empyema of right maxillary antrum and spreading osteitis of right maxilla, zygoma, temporal and frontal bones. Extensive craniectomy. Aspiration of abscess. Eventual extirpation of abscess. Recovery.

B.F., male aet. 30 (R.I. 12120) was admitted to the Radcliffe Infirmary on 13th November, 1940. Except for long-standing nasal obstruction and catarrh, he had been in good health until 19th September, 1940, when he had an infected molar tooth extracted from the right upper jaw, under local anaesthesia. The dentist reported that the extraction was very difficult and that a fragment of the floor of the antrum came away with the roots of the tooth. On the following day the right side of the face was swollen and he appeared to be generally ill. The medical record of the ensuing three or four weeks is incomplete, but it seems that by 25th September he was gravely ill, with a generalised infection which was treated with M. & B. 693 with no effect, but M. & B. 760 brought about a definite improvement. The inflammation seemed to settle down, but during the first week of October he had a right-sided pneumonia. This too resolved, and he gradually improved to the point of being able to get up in his room and to dictate letters. On 4th November there was another rise of temperature, he began to complain of headache, and he became very drowsy. On 12th November there was a period of coma, lasting for several hours, from which he was roused by the administration of hypertonic solution.

On admission he was very drowsy and inattentive, but he could be roused to cooperate in a modified examination. Pulse, temperature, and respirations were normal. There was a little oedema of the right side of the face, and of the right temporal region, extending up to

the right side of the forehead. He complained of pain on pressure over the zygoma, temporal muscle, and the right lateral border of the frontal bone. The neurological examination revealed bilateral papilloedema, a right external rectus palsy, definite weakness of the left side of the face, left arm and leg, brisk tendon reflexes in the left limb, and bilateral extensor plantar responses. There seemed to be no sensory defect, nor any defect in the visual fields as tested by confrontation. X-rays of the skull showed only impaired translucency of the right antrum, and Mr. R.G. Macbeth considered that his illness had begun with an acute exacerbation of a chronic infection of the antrum. He did an exploratory puncture on 14th November and found foetid, flocculent pus. A lumbar puncture on the same day showed that the pressure was 300 mm., the fluid was clear and colourless, containing 60 mgm. protein and 8 lymphocytes.

The superficial signs of inflammation in the right side of the face and right temporo-frontal region suggested that there was infection of the underlying bone; the neurological signs and the spinal fluid pointed to a brain abscess as well.

On 16th November, 1940, a ventriculogram was done (Fig. 48) and this indicated an abscess at the tip of the right temporal lobe. A right frontal flap was reflected on the same day. The temporal muscle was oedematous and throughout it there were small pockets of pus and areas of necrosis. These were excised. The underlying squamous temporal bone was infected, and the inflammation extended to the adjacent antero-lateral part of the frontal bone, the frontal process of the zygomatic bone, the lateral end of the sphenoidal ridge, and the lateral wall of the orbit. All of the infected bone was removed.

Fig. 48 overleaf. Case 19.

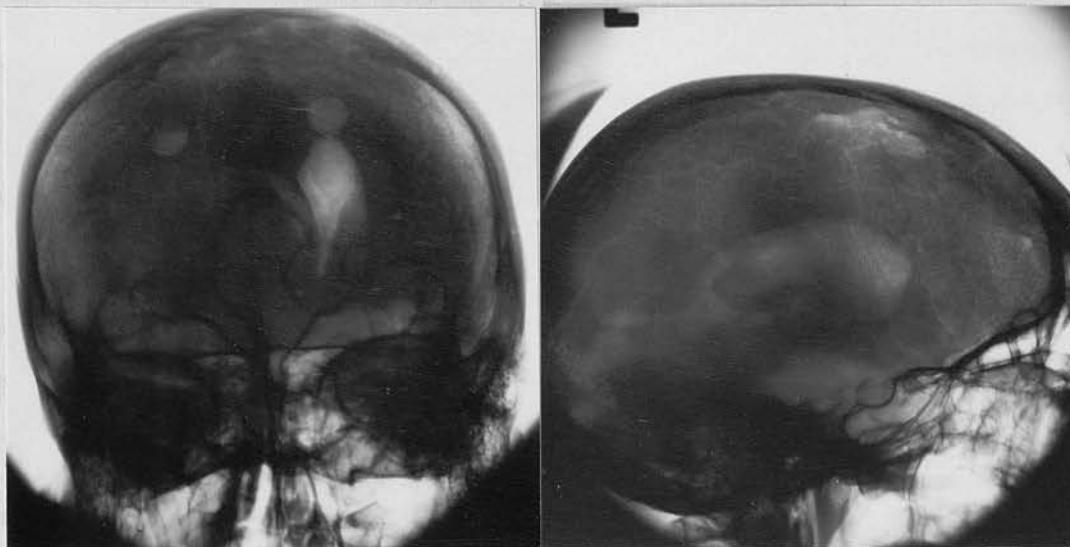
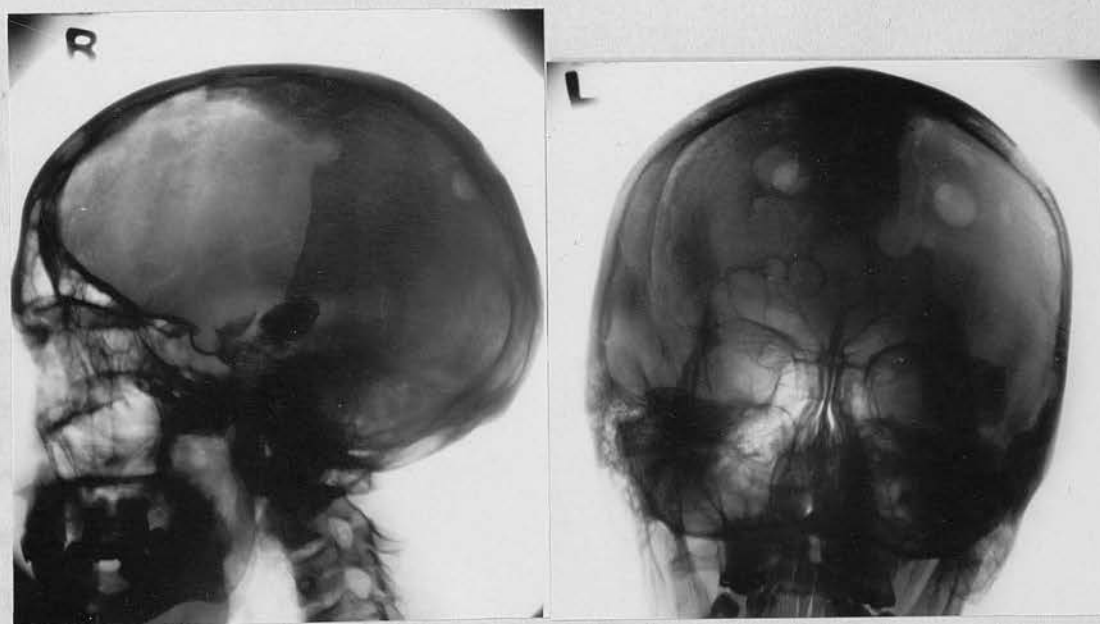
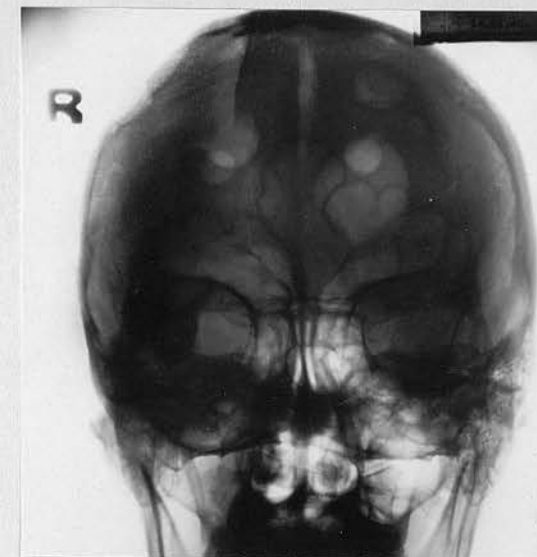


Fig. 48. Case 19 - Ventriculogram 16:xi:40, showing displacement of whole ventricular system to left side, and obliteration of right temporal horn.



Pyograms 22:xi:40 showing extent of craniectomy and outline of abscess in anterior part of right temporal lobe.



Pyogram taken after initial aspiration. Compare the size of the abscess with the appearance six days later seen in the lower left plates.



Pyogram 31:xii:40. Note that the abscess has not increased in size. The worsening of the clinical condition suggested that there were other loculi which were not filled with thorotrast. See text, page 237.

X There was a pod of granulation tissue covering the dura over the lateral aspect of the temporal pole, and from this beads of pus exuded. The dura elsewhere looked normal. A brain needle was passed through a nick in healthy dura posterior to the granulation tissue, and at a depth of 1 cm. in the temporal lobe the needle was felt to slip into an abscess from which 8 c.c. of thin yellow pus were aspirated. From this, as from the bone and temporal muscle, staphylococcus aureus was cultured. One c.c. of thorotrast was instilled into the cavity (Fig. 48).

The post-operative course was very stormy. The wound healed normally and remained intact, but he continued to be stuporose and incontinent, and although the temperature was always normal he had frequent rigors and bouts of profuse sweating. On 18th November there was a complete right ophthalmoplegia, and it was thought that he had a thrombosis of the cavernous sinus. This cleared up in three days, however. Repeated spinal fluid examination showed a slight increase in protein (60 mgm.) and 1-3 cells. Blood cultures were sterile. On 29th November, 2 weeks after operation, he was in such a low state generally that his life was despaired of. On this day intensive treatment with sulphathiazole was begun, and from that time he began to improve: he became more alert, the rigors ceased, the decompression at the site of the craniectomy was quite soft, and the papilloedema subsided. The left hemiparesis persisted, however, and as he became more alert it was possible to demonstrate an upper quadrant defect in the left homonymous field of vision.

A ventriculogram on 1st January, 1941, revealed considerable bilateral ventricular dilatation and upward displacement of the anterior

part of the right temporal bone (Fig. 48). On 6th January, when it was reckoned that the abscess was about 12 weeks old, the right frontal scalp flap was re-elevated and the abscess was dissected out of the temporal lobe. It had a thick wall and several loculi, and proved to be larger than the pyogram indicated, as it occupied the anterior half of the temporal lobe. It was adherent to the dura at the site of the granulation tissue seen at the first operation.

Improvement after this operation was very gradual. The wound healed normally and the spinal fluid was normal (45 mgm. protein, 0 cells) by 9th February. The defect in the left upper quadrant of the visual fields disappeared, and acuity was normal in each eye. The weakness of the left side of the face and left upper limb cleared up fairly quickly, but the left lower limb was very weak and spastic and was held immobile in flexion, abduction and external rotation at the hip joint. He complained of great pain on movement of the joint, and although there were no radiological abnormalities it was thought that he probably had a low-grade septic arthritis. Aspiration was negative, however, and the leg was finally straightened out by gradual traction.

Even more gradual than his physical recovery was the improvement in his mental state. He continued to be very drowsy and incontinent, but when roused he passed formal intelligence tests with the marks of a superior adult. Apathy, indolence and incontinence were still marked when he was sent home to complete his convalescence on 16th March, 1941. Improvement continued, but it was not possible to arrange another examination until 21st August, 1941. At this time he said he was in his normal good health, and the only demonstrable neurological abnormality was a slight residual weakness of the left lower limb

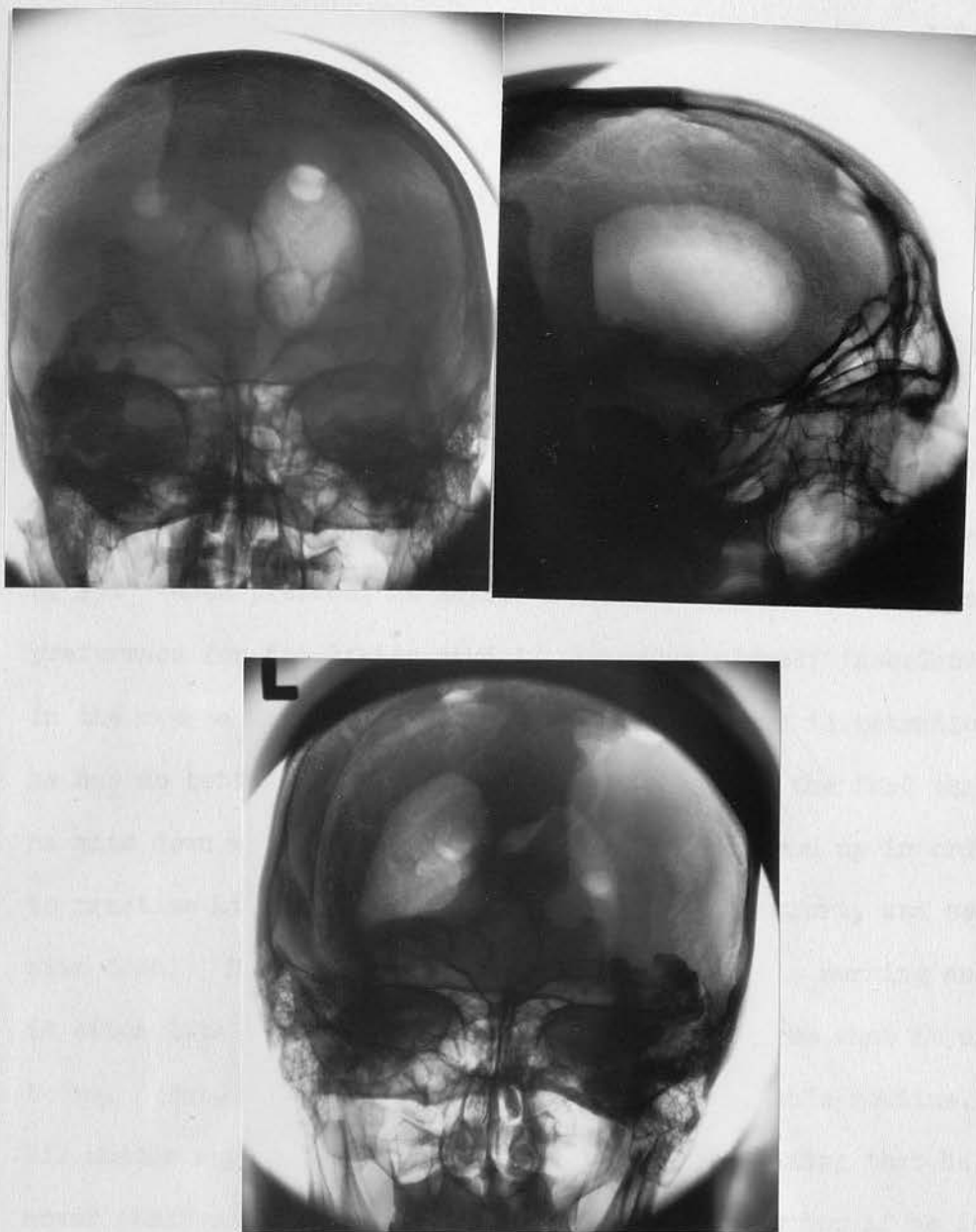


Fig. 48 a . Case 19 - Ventriculogram 1:i:41, showing dilatation of ventricles, displacement to left side, and obliteration of right temporal horn.

which did not inconvenience him. The decompression was indrawn, but it was covered by thick curly hair and was no unsightly (Fig. 49). On a superficial level his intelligence and behaviour seemed to be perfectly normal. Dr. Erich Guttman made a detailed psychiatric study and reported as follows:

"The patient himself has no mental symptoms to complain of. His memory and concentration are satisfactory; he is cheerful as ever. His interests have not changed or diminished. He greets his friends and talks to them as he always did. They have noticed no change in him, at least they have never commented on it. When pressed, he says, 'I am not very brisk. I have a preference for the easier ways.' He calls himself 'indolent' in the course of the conversation. Asked for an illustration, he has no better example of his 'indolence' than the fact that he sits down when dressing, whereas he should stand up in order to practise his leg. But that needs too much effort, and so he sits down. He is a bit slow in getting up in the morning and is often late for breakfast; that is different from what it used to be. But, of course, this was the business man's routine. His mother says, 'You have no sense of time', meaning that he is never punctual, and he admits that he does not bother if he is late. (I suggested yesterday seeing him at 9-30 today. He asked if 10 would be all right; it meant finishing his breakfast. Finally he was 45 minutes late.) During the interview he showed no sign of 'laziness' - he cooperated very readily, showed much interest in the tests and tried hard; even in tests which were beyond his capacity he did not easily give in. (This was fundamentally

Fig. 49 overleaf. Case 19 -

Fig. 49. Case 19 - Post-operative photograph,
showing the slight depression at the site of
the incision caused by the use of the
scalpel.

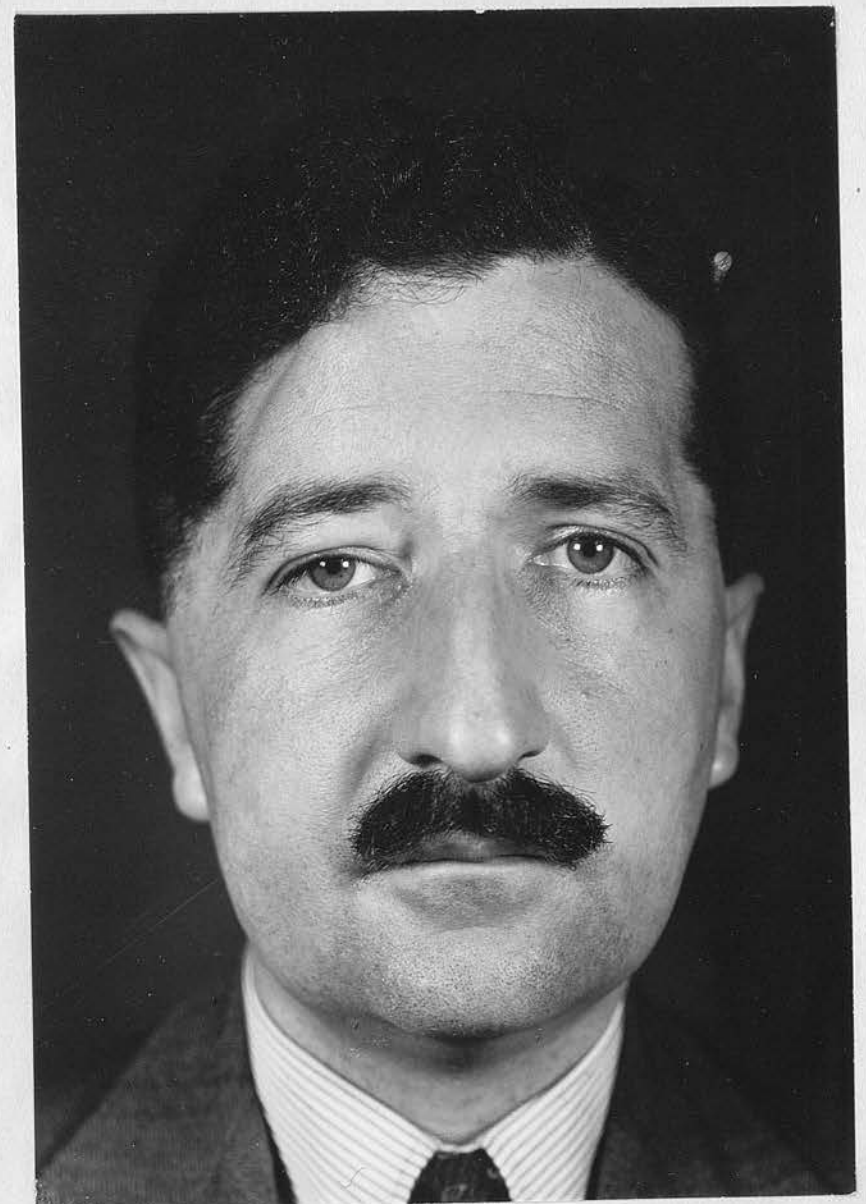


Fig. 49. Case 19 - Post-operative photographs,
showing the slight depression at the site of
the craniectomy covered by hair and hardly
noticeable.

different from last time.) His behaviour was ~~all~~ right; he tended perhaps to comment more freely on the procedure than invited, and he yawned without much restraint after an hour's testing.

"Intellectual Level: Both in verbal and non-verbal tests he seemed definitely above average (Wechsler I.Q. 130; Raven 48 items+). This shows that the Vocabulary Test gave us, during his mental illness, a fair estimate of his potential intellectual level. It was in these tests that he showed today not only his capacity but also his willingness to think and to reason. Retrospectively, it confirms that his intellectual faculties were not lost, but only inaccessible at that time, or not working owing to lack of driving power.

"Efficiency: Counting, calculation, days and months, forward and reversed, he does quickly and correctly.

"Memory: 8 digits forward and 5 backwards easily. Rey-Davis learning curve optimal. His recollection of his mental episode is very patchy, but his lack of interest and initiative stands out in his own memory ('I did not care').

"Summary: The only residual symptom appears to be a slight lack of initiative as evidenced mainly by his self-description."

He returned to his former work in an insurance office on 29th October, 1941, and has continued without interruption in the $2\frac{1}{2}$ years since then. Periodic examinations have revealed some abnormalities which appear to be more in the moral than intellectual sphere: he has little regard for punctuality and is often late for business and social appointments, and does not seem to be upset by thereby inconveniencing other people. His mother and sister noticed a certain lack of regard

for their feelings. He paid attention to insignificant symptoms, and would often drop in to hospital out-patient departments to have some kind of investigation done, and he became something of a bore in detailing his symptoms. To those who had not known him before his illness these peculiarities would probably have passed unnoticed, but they were very real to his family and intimate friends.

Case 20. Left temporal abscess due to cavernous sinus thrombosis. Chronic staphylococcal meningitis. Failure of diagnosis. Death.

V.R., female aet. 29 (R.I. 26160) was admitted to the Radcliffe Infirmary on 17th September, 1942. She had been in good health until the end of May, 1942, when two or three small boils appeared on her face. One of these was squeezed and another lanced within four days of the onset. On the following day she had a very severe headache, and both eyes were swollen. These symptoms led to her admission to the City General Hospital, Leicester, on 2nd June, 1942. Dr. A.P.M. Page reported that on admission there was a healing boil on each cheek, both eyes were protuberant, and there was a marked swelling of the lids and conjunctivae. Her neck was stiff and Kernig's sign was present in both legs. Lumbar puncture revealed turbid fluid, with a marked neutrophil reaction, and staphylococcus aureus was grown on culture. This organism was also grown in blood culture. It seemed clear that she was suffering from staphylococcus bacteraemia, with cavernous sinus thrombosis and meningitis.

She was treated energetically with sulphathiazole, and there was dramatic improvement. By 16th June, the signs of meningitis had cleared up and the spinal fluid contained only 56 cells and was sterile on culture. She was afebrile, and the proptosis had diminished. During the acute phase of her illness it was noticed that she had a slight right hemiparesis and a mild aphasia.

There were three subsequent recurrences of the meningitis, but each was dealt with by sulphathiazole, and her general condition improved. The hemiplegia and aphasia became more marked as time went

on, and it was thought that these signs were due to an abscess in the left hemisphere.

On admission, $3\frac{1}{2}$ months after the onset of the infection, she looked reasonably well, considering the protracted illness. Her temperature was 99° , pulse rate 100/min., and respirations were normal. There was a well-marked global aphasia, affecting all aspects of the language function, but she was alert and intelligent, so that it was possible to converse with her fairly easily. There were no superficial signs of sepsis, but both eyeballs were a little prominent, and a slight bilateral ptosis gave her a rather sleepy look. Both optic fundi were congested, but the papilloedema was not of measurable degree. The visual fields showed an incongruous right homonymous hemianopia of the type seen in lesions of the optic tract. The external rectus muscle was weak on both sides, but the ocular movements otherwise were normal. The right side of the face was weaker than the left in emotional and voluntary movement, and it shared in a slight right hemiplegia affecting the arm and leg equally. The right limbs were slightly hypertonic and there was a constant tremor which was exaggerated in purposive movements. The tendon reflexes were slightly brisker on the right side, and the right plantar response was extensor. Although cutaneous stimuli were felt equally well on both sides of the body, postural sense, stereognosis, and two-point discrimination were defective in the right arm and leg. X-rays of the skull were normal. On lumbar puncture the pressure was 270 mm., the fluid was clear and slightly yellow. It contained 240 mgm. protein, 108 cells (62% neutrophils) and was sterile on culture.

The clinical evidence thus favoured a lesion in the internal capsule,

and from the history an abscess seemed likely. On the other hand, she seemed to be very well to be harbouring an abscess, and headache had not figured largely in her story, except during the meningitic phases. An alternative which suggested itself was that she was suffering from a chronic low-grade meningitis, and the hemiparesis and aphasia were due to cerebral thrombophlebitis.

Ventriculography was performed on 30th September, 1942. Both ventricles were capacious and the pressure was atmospheric. The pictures (Fig. 4) showed the ventricular dilatation, doubtless the sequel of the meningitis, and very slight displacement to the right side. No filling defect could be seen, and it was thought that the displacement was due to oedema of the left hemisphere and to accumulation of cerebrospinal fluid in the subarachnoid space over the hemisphere rather than to an abscess. Although the ventricular fluid was clear and colourless, it contained an excess of protein (50 mgm.) and cells (26, of which 64% were lymphocytes), suggesting that there was inflammatory process within the ventricles too, and no indication for surgical treatment was recognised. She was sent back to the City General Hospital, Leicester, where she remained in much the same state until 12th November, 1942, when she died suddenly.

Dr. G.M.Dabrashian removed the brain and found that the cavernous sinus seemed to be obliterated by fibrous adhesions on the left side, and that there was a thrombosed vein passing from the cavernous sinus to the pole of the left temporal lobe. The convolutions on the inferior surface of the left temporal lobe were flattened, and when the brain was being removed an abscess in the medial border of the temporal lobe was ruptured. When the brain was sectioned (Fig.50)



Fig. 50. Case 20 - Ventriculogram showing internal hydrocephalus, and very slight displacement to right of third ventricle. This displacement is due to the deep-seated abscess seen in the plate below. The ventricular dilatation was doubtless due to the meningitis which occurred in the early stages of the illness.



a thick-walled abscess was found in the medial part of the temporal lobe at the genu of the internal capsule. There was a swollen and more recent abscess medial to this one, extending to within 0.3 cm. of the meninges over the uncus; it was this one which ruptured during the removal of the brain. The post-central gyrus and the insula were atrophic, and there was considerable subcortical necrosis, suggesting the effects of venous thrombosis in the vicinity.

Case 17. Right frontal abscess (*staphylococcus aureus*) following removal of right frontal meningioma. Aspiration. Recovery.

L.A., male aet. 41 (R.I. 9457) was admitted to the Radcliffe Infirmary on 11th July, 1940, complaining of severe headache and frequent bouts of double vision. The clinical and radiological investigations pointed to a neoplasm in the right frontal lobe, and at operation on 23rd July, 1940, a parasagittal meningioma was removed. Convalescence was uneventful until the eighth day after operation, when there was a rise of temperature to 101° and the scalp flap became red and swollen along the middle limb of the incision. On the following day there was some sero-purulent discharge from the wound from which *staphylococcus aureus* was grown. Although the wound continued to discharge pus, his general condition was satisfactory. He had no headache, felt well in himself, and was getting up and helping with the ward work. There were no neurological abnormalities. He was treated from the outset with sulphathiazole by mouth and frequent antiseptic dressings, but there was no lessening of the discharge, and by the end of eight weeks there were definite signs of infection of the bone flap (Fig. 46). Accordingly, on 4th October, 1940, the scalp flap was re-elevated and the medial half of the bone flap was excised. There was a mass of extradural granulation tissue, and through a gap in the dura over the cavity from which the tumour had been removed there was a small cerebral hernia protruding. The scalp flap was re-sutured, a drainage tube being led out through a stab wound behind the posterior limb of the incision. Although there was no further discharge, the middle limb of the scalp wound failed to heal, and the cerebral hernia